

Clinical Methods in CARDIOLOGY



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Foreword

These are exciting times for cardiology! Dramatic breakthroughs in basic research, exciting results of major clinical trials, and new diagnostic and therapeutic devices have revolutionized cardiovascular care worldwide. Knowledge and procedures previously available only in large research centers are now available in local and community hospitals. This rapid diffusion of information and technology is coming just in the nick of time, as cardiac and vascular disease is destined to become one of the major public health problems worldwide in this millennium. One of the unfortunate byproducts of the rapid maturation of many developing countries is the development of additional cardiac risk factors, and the emergence of a mini-epidemic of cardiovascular diseases, particularly coronary artery disease.

It is in this milieu that B Soma Raju's *Clinical Methods in Cardiology* is so timely and so appropriate. It addresses two major issues prompted by the rapid dissemination of medical information, procedures and technology throughout the globe: the need for practitioners to understand the fundamentals of the history and physical examination, rather than rely blindly only on technology; and the need to be aware of the unique patient-care issues of different regions of the globe. This textbook addresses both issues in an outstanding manner. It is a distillation of Dr Soma Raju's clinical approach and experience as one of the most respected cardiologists in India. The book is a private tutorial outlining one master clinician's approach to the cardiac history and physical examination. His thesis (with which I agree) is that the clinical evaluation of the patient forms the foundation upon which the management of the patient is built. Without such a firm basis, optimal treatment of the patient is not possible.

I have had the pleasure and honor of working with Dr Soma Raju over the last decade on two collaborative research projects studying the optimal treatment of rheumatic mitral stenosis that were a joint effort between my institution and his. In that period I have come to cherish my times in India, and my association with him. He is the best bedside clinical diagnostic cardiologist I have ever seen, and his enormous wealth of clinical experience is well reflected in *Clinical Methods in Cardiology*. I believe that this textbook is destined to become the definitive text from the subcontinent on the clinical approach to the cardiac patient.

Joshua Wynne

MD

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Preface

Physical examination and history continue to play a pivotal role in the evaluation and management of patients with cardiovascular disease. Currently there is an increasing tendency to rely on laboratory tests with the assumption that these methods are more 'objective'. The following case summary illustrates this trend.

A 45 year old male hypertensive went for annual medical examination. The exercise test was interpreted as 'strongly positive for myocardial ischemia'. Coronary angiography was done. During the angiogram, the left coronary catheter repeatedly 'damped' (Fig 1); this was suspected to indicate left main ostial stenosis particularly on the background of a 'strongly positive' exercise test.

The angiogram was interpreted as 'consistent' with left main ostial stenosis though the quality of the angiographic film was far from satisfactory. The cardiologist argued that the strongly positive exercise test explains the angiographic finding and the The cardiovascular surgeon readily agreed with the interpretation. Emergency coronary bypass surgery was advised. The patient was taken aback by the rapid sequence of events as he had never had any symptoms and was exercising regularly for more than an hour daily with no difficulty. He was warned of the danger delaying surgery in this setting and there was imminent danger to his life. The patient opted for a discharge from the hospital and sought another opinion. The review of the angiogram revealed that the quality of the film was poor and no conclusions could be drawn from it. As the patient was a hypertensive, an echocardiogram was done which showed concentric left ventricular hypertrophy. This explained his false positive 'strongly positive' exercise test. Angiogram was repeated with a 3.5 Judkin's left coronary catheter. There was 'damping' of pressure

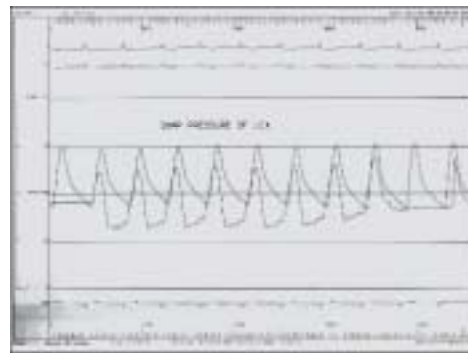


Fig 1



Fig 2

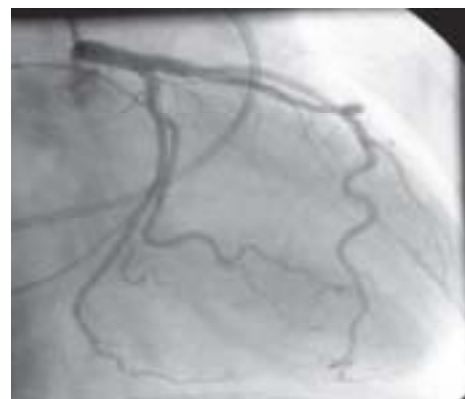


Fig 3

from the coronary catheter as was observed in the earlier investigation. But the angiographer felt that there was a back flow during the coronary injection which could be easily made out because of superior angiographic system. A Judkin's 4 curve left coronary catheter was used to test the 'false positive' or pseudo-damping. There was no damping this time and the angiogram was interpreted as normal. The 3.5 curve damped because the catheter tip was abutting en face against the roof of the left main artery. The patient was discharged next day to resume his normal activities and exercises. He was in excellent health 6 years after the angiogram.

This patient almost had an unnecessary bypass surgery based on 'objective' tests. Without 'subjective' information, hardly any decision can be made in the care of patients. It should be realized that objective laboratory tests are not necessarily superior to subjective clinical methods. The 'objectivity' is particularly misleading because of uncritical and almost slavish over-reliance on anything that is based on sophisticated technology.

In the assessment of stenotic valve disease, cardiac catheterization and hemodynamic calculation of valve areas is considered the gold standard amongst the presently available methods. The following table from the study of Defillippo et al illustrates the limitations of even this method.

Table: Limitations of cardiac catheterization—patients initially diagnosed to have aortic stenosis

Patient	Ao BP (mm Hg)	Flt. Vel. (m/s)		PPG (mm Hg)		MPG (mm Hg)		AVA (cm ²)		Valve Resist. (dynes/cm ²)		AVR	1-Year (f/u)
		Bs	Dob.	Bs	Dob.	Bs	Dob.	Bs	Dob.	Bs	Dob.		
1	92/68	3.0	3.6	36	52	17	29	0.7	0.7	152	236	+	A & W
2	112/60	3.8	4.5	58	81	27	45	0.7	0.8	196	211	0	Dead
3	—	3.2	3.9	41	61	24	39	0.8	0.7	196	330	0	Alive
4	109/69	3.6	4.5	52	81	27	44	0.6	0.6	246	323	+	A & W
5	90/62	3.4	4.3	46	74	25	43	0.7	0.8	169	211	+	Dead
6	92/60	3.7	4.9	55	96	30	60	0.6	0.7	254	346	+	A & W
7	—	2.3	3.4	21	46	12	26	0.8	0.9	116	168	0	—
8	—	2.8	3.4	31	46	19	27	0.9	1.2	119	114	0	A & W
9	176/90	2.9	3.5	34	49	19	25	0.8	1.1	145	126	0	Dead
10	104/64	2.7	2.7	29	29	19	18	0.8	1.1	145	99	0	A & W
11	108/48	2.5	2.7	25	29	15	20	0.7	1.1	156	118	0	A & W
12	165/85	2.8	3.2	31	41	19	21	0.8	1.1	155	117	0	A & W
13	124/80	2.9	3.3	34	44	19	25	0.9	1.1	158	123	0	CHF
14	120/68	3.6	3.7	52	55	29	34	0.6	0.7	245	261	0	Dead
15	140/98	2.4	2.6	23	27	13	16	0.9	1.0	115	123	0	Dead
16	150/80	3.3	3.3	44	44	26	28	0.8	0.9	182	172	BVP	Dead
17	—	3.1	3.2	38	41	15	17	0.9	0.8	113	118	0	CHF
18	175/69	3.4	3.5	46	49	24	27	0.6	0.7	258	206	0	CHF

Patients were initially thought to have severe aortic stenosis because valve areas were calculated at low cardiac outputs. Hemodynamic manipulation by dobutamine or valve inspection proved the initial assessment to be incorrect (Defilippo et al 1995, *Am J Cardiol* 75: 191–94).

The mistakes in the measurement of intracardiac pressures and cardiac output are common in the cardiac catheterization laboratories all over the world. Many laboratories do not even have adequate hemodynamic measurement and recording systems.

The 'objective tests' when improperly done without adequate quality control are particularly less reliable and even dangerously misleading. The decisions for definitive procedures like cardiac surgery and catheter interventions are made from the 'objective' information provided by them. Medicine deals with human beings who are subjects and not objects on *which* tests are done and procedures are carried out.

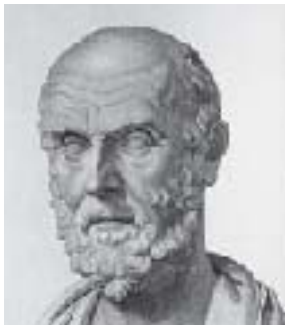
Though there are books on physical examination of the heart by well-known authors, I decided to put to print my way of looking at things. I fully realize that there are many ways of looking at things and hence the need for more of us to communicate by writing. This book is written with a firm conviction that physical examination of a patient is a way of thinking and not merely a way of doing. It plays a vital role in decision making: both diagnostic and therapeutic. I hope this will guide the medical student, the postgraduate, the cardiologist in training, the practising physician, the medical teacher and the practising cardiologist.

When there is a discrepancy between the ground and the map, the ground is usually right.

Field Marshal Erwin Rommel

The map is the angiogram and the ground is the patient.

Note to the reader



Hippocrates, the first Greek to challenge the notion that disease was punishment sent from the gods, discovered the connection between human disease and poor environmental conditions. Considered to be the father of medicine, his ability to make accurate clinical observations led him to the concept of preventive medicine.

THE OATH OF HIPPOCRATES (MODIFIED)

I swear to fulfill to the best of my ability and judgment, this covenant.

I will respect the hard-won scientific gains of those physicians in whose steps I walk, and gladly share such knowledge as is mine with those who are to follow.

I will apply for the benefit of the sick, all measures which are required, avoiding those twin traps of overtreatment and therapeutic nihilism.

I will remember that there is art to medicine as well as science, and that warmth, sympathy and understanding may outweigh the surgeon's knife or the chemist's drug.

I will not be ashamed to say "I know not," nor will I fail to call in my colleagues when the skills of another are needed for a patient's recovery.

I will respect the privacy of my patients, for their problems are not disclosed to me that the world may know. Most especially must I tread with care in matters of life and death. If it is given me to save a life, all thanks. But it may also be within my power to take a life; this awesome responsibility must be faced with great humbleness and awareness of my own frailty. Above all, I must not play at being God.

I will remember that I do not treat a fever chart, a cancerous growth, but a sick human being, whose illness may affect the person's family and economic stability. My responsibility includes these related problems, if I am to care adequately for the sick.

I will prevent disease whenever I can, for prevention is preferable to cure.

I will remember that I remain a member of society, with special obligations to all my fellow human beings, those sound of mind and body as well as the infirm.

If I do not violate this oath, may I enjoy life and art, respected while I live and remembered with affection thereafter. May I always act so as to preserve the finest traditions of my calling and may I long experience the joy of healing those who seek my help.

translated by Louis Lasanga (1964) Academic Dean, Tufts University

The following paragraphs are excerpts taken from the Presidential address of Professor Douglas Zipes at the plenary session of the American College of Cardiology in March 2002:

The way medicine is practised today raises many questions about where our concerns and our responsibilities must lie in this world. The questions are many and the answers difficult. But one answer, particularly for us as physicians, stands out above all the rest. We now know that our concerns can no longer be limited to a personal agenda, to a regional interest, or to any single corner of any particular market. We now know that the proper sphere of influence for every responsible man and woman is this entire fragile sphere called earth. Castes and communities, religious fanaticism, regional parochialism, the and errant politicians fade into the mist of dreams when held up to the pleading realities affecting our world. It has become blindingly clear that our concerns must encompass the relief of misery wherever we find it. And that means that we, as physicians and cardiologists, must expand our vision to include all of humankind.

Our world has been likened to a beautiful book that is of little use to those who cannot read. And in a similar sense, our knowledge as physicians is a fine and priceless asset whose value is vastly diminished unless it can be taught, disseminated, and practised throughout the world. In Peru, Calcutta, and Afghanistan.

I have written about it in one of my president's pages, and I like to call it a patient in a box. It is designed to teach physicians at all stages of their careers how to use new medical devices and perform new procedures.

It will allow trainees to practice new techniques without fear of harming patients. In fact, in the future a certain number of practice hours logged on a simulator will probably become required before performing a procedure on a patient.

It will help us to work and train as a team, along with our nurses, technicians, and physician colleagues. Dealing with rare or infrequent complications, such as cardiac perforation and tamponade, can be practised many times on the simulator, so that when it actually happens, the cath team can respond like a crack drill squad.

In the near future, from the first venopuncture that a medical student performs to a complex angioplasty in the last year of cardiology fellowship training, procedures will be taught in such virtual reality settings. Perhaps, also in the future, low volume operators will be able to make up for lack of patient numbers by documented hours spent practising on a simulator.

I submit to you that virtual reality is an unquestionable part of our educational future, and it is a means through which we can spread knowledge all over the world. It will change forever how we teach, test, and treat.

We must educate the public of the wrong doings and unethical practices they are subjected to.

What else can we do, as individual clinicians and as a College, to improve the quality of the care that we deliver? How can we demonstrate that quality to regulatory agencies, third-party payers, and, most importantly, to our patients and their loved ones?

In searching for an answer to these questions, we must first look to the nature of our relationship with the society in which we live.

We are social animals with a need to coexist in the company of others, and to interact with them. It

seems to me that the moral glue that binds us together comes in large measure from our accountability to those others.

For each of us is accountable in some way to someone . . . a husband to his wife, a parent to a child, a physician to a patient. We know this and live by it.

And in these interactions, we think of ourselves, for the most part, as honorable men and women with standards that are an unshakable part of our accountability to society. Those standards are founded on an underlying knowledge of what is right and what is wrong.

And, as physicians, we think of ourselves as knowledgeable and competent professionals with a distinct understanding of what is right for our patients, and what is not. We know this with complete confidence. And rarely, if ever, do we find the need to question it.

And, in the most immediate sense, that accountability applies directly to the vulnerable state of the patients whom we treat.

I like to think that good health is when your body does not talk to you, when it is silent. You are largely unaware of your body when you are healthy. You don't consciously think about having an arm, a head, or a stomach.

But you know very well that you have a back when it aches.

And you know very well that you have a heart when your chest hurts.

It is then that your body talks to you.

We physicians tend to see people when they are most aware of their bodies, when their bodies are talking to them a lot. Which means that we see them when they are at their most vulnerable.

We see them when they are undressed in every way—physically, emotionally, and spiritually. To see them so places us in a position of enormous privilege and responsibility.

Because we are all the same when we are naked.

The wise and the foolish.

The mighty and the weak.

The wealthy and the will be gone.

All the same . . . all vulnerable.

And it is this vulnerability that endows the physician with stunning privilege, and an equally stunning responsibility.

For it is our privilege to shield our patients when they are bare and without defenses.

And to listen to the voice of the patient, not the voice of the disease.

And to clothe them not only with health but also with the ability to thrive once they have left our care.

And to be their friend, their counselor, their trusted advisor.

These are concerns that apply to every physician in the world. It has nothing to do with national borders, with ethnicity, or with religious affiliation.

It has everything to do with the framework of humankind that needs our constant support, not just as physicians but as men and women of sensibility and conscience.

It has everything to do with the achievement and exportation of excellence and it has everything to do with the Hippocratic Oath, which as a profession we swear.

It has everything to do with putting the patient first, above our own interests.

And we have a great deal to give, a great deal to share with the world.

We must always bear in mind that excellence is elastic. It knows no limits. And it must be maintained not only by the preservation of the best of the past, but by the brilliance of the future, by the need to dare, and by the willingness to embrace both innovation and experimentation, and to stay abreast of these advances.

For excellence is a reflection of spiritual wealth, and the exportation of that wealth is a notion that we cherish. And to me that notion is what defines the difference between a profession and a calling.

Because a profession is something that you train to do. A profession is something that you can change; it has an impermanence about it. A profession is something that you are likely to find routine in later years.

A calling, on the other hand, is something that captures you, entrances and embraces you, and keeps you enchanted for the rest of your life.

You see, those who have the calling must be healers by conviction, not simply by virtue of a medical degree.

We become healers when the identifiable purpose of our lives is forever bound up with the relief of suffering, with the forestalling of death or its embrace when the time has come, and with the creation of environments where our patients can flourish.

We become healers when the relationship between our patients and us is a covenant of faith, not a business contract; an article of trust, not simply a fee for services.

We become healers when we come to understand that healing is hard work, for both the patient and the physician, that the amount of health that we can actually promote is relatively small when weighed on the scale of human mortality.

And we become healers when we ignore that scale and fight for every inch of health, against the odds, as if embedded in our fingertips is the ability to create that body that does not talk to you.

These are the sort of healers that our profession cries out for—men and women who are willing to labor in the trenches.

Who are willing to treat all patients equally.

Who are willing to touch what others see as untouchable.

Who are willing to strive for nothing less than the promise of blood, toil, sweat, and tears in return for nothing more than the privilege of healing and saving.

To treat each day as if it were your last, and each patient as if he or she were your first.

“We should be ashamed to die until we have won some victory for humanity.”

Saving one life helps save that humanity.

Modified from the Presidential Address of Dr. Douglas Zipes
at American College of Cardiology, March 2002

Abbreviations

ABG	Arterial blood gas	HOCM	Hypertrophic obstructive cardiomyopathy
ACG	Apex cardiogram	HTN	Hypertension
AF	Atrial fibrillation	IABP	Intra-aortic balloon pump
AIDS	Acquired immunodeficiency syndrome	ICCU	Intensive coronary care unit
AM	Acute margin	JVP	Jugular venous pressure
AMI	Acute myocardial infarction	L-TGA	L-transposition of great arteries
AR	Aortic regurgitation	LA	Left atrium
AS	Aortic stenosis	LAD	Left anterior descending artery
ASD	Atrial septal defect	LBBB	Left bundle branch block
AV	Atrioventricular	LCA	Left coronary artery
BP	Blood pressure	LCX	Left circumflex
CABGS	Coronary artery bypass graft surgery	LIMA	Left internal mammary artery
CAD	Coronary artery disease	LLSB	Left lower sternal border
CASS	Coronary artery surgery study	LMCA	Left main coronary artery
CCF	Congestive cardiac failure	LPSI	Left parasternal impulse
CCU	Coronary care unit	LSB	Left sternal border
CCS	Canadian Cardiovascular Society	LV	Left ventricle
CHB	Complete heart block	LVEDP	Left ventricular end diastolic pressure
CI	Cardiac index	LVF	Left ventricular failure
CMV	Closed mitral valvotomy	LVH	Left ventricular hypertrophy
CP	Constrictive pericarditis	LV-RA	Left ventricle to right atrium
CT	Computerised tomography	defect	defect
D1	First diagonal	MI	Myocardial infarction
D2	Second diagonal	MR	Mitral regurgitation
D-TGA	Dextro-transposition of great arteries	MRI	Magnetic resonance imaging
DKA	Diabetic ketoacidosis	MS	Mitral stenosis
DW	Dicrotic wave	MVO ₂	Myocardial oxygen consumption
ECG	Electrocardiogram	MVP	Mitral valve prolapse
EMF	Endomyocardial fibrosis	MVR	Mitral valve replacement
ESM	Ejection systolic murmur	NYHA	New York Heart Association
GI	Gastrointestinal	OM	Obtuse marginal
HCM	Hypertrophic cardiomyopathy	OMV	Open mitral valvotomy
		PA	Pulmonary artery

PAH	Pulmonary arterial hypertension	RVF	Right ventricular failure
PAW	Pulmonary artery wedge	RVH	Right ventricular hypertrophy
PBMV	Percutaneous balloon mitral valvoplasty	RVOT	Right ventricular outflow tract
PCV	Packed cell volume	S1	First heart sound
PCWP	Pulmonary capillary wedge pressure	S2	Second heart sound
PDA	Patent ductus arteriosus	S3	Third heart sound
PD artery	Posterior descending artery	S4	Fourth heart sound
PLVB	Posterior left ventricular branch	SAH	Subarachnoid hemorrhage
PMC	Premature mitral closure	SAS	Specific activity scale
PMD	Papillary muscle dysfunction	SSS	Sick sinus syndrome
PND	Paroxysmal nocturnal dyspnea	STK	Streptokinase
PPV	Positive pressure ventilation	SVT	Supraventricular tachycardia
PR	Pulmonary regurgitation	TAPVC	Total anomalous pulmonary venous connection
PS	Pulmonary stenosis	TGA	Transposition of great arteries
PTCA	Percutaneous transluminal coronary angioplasty	TIA	Transient ischemic attack
PVH	Pulmonary venous hypertension	TR	Tricuspid regurgitation
RA	Right atrium	TS	Tricuspid stenosis
RBBB	Right bundle branch block	TVD	Tricuspid valve disease
RCA	Right coronary artery	VAS	Visual analogue scale
RHD	Rheumatic heart disease	VBI	Vertebro basilar insufficiency
RIMA	Right internal mammary artery	VSD	Ventricular septal defect
RV	Right ventricle	VT	Ventricular tachycardia
		WPW	Wolff–Parkinson–White syndrome

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1 The Science and Art of Medicine

There are qualities beyond pure medical competence that patients need and look for in their physicians. They want reassurance. They want to be looked after and not just looked over. They want to be listened to. They want to feel that it makes a difference to the physician.

Norman Cousins

The importance of becoming a doctor is not fully realized by the majority of students at the time of joining the medical course. Most of us took up medicine because that was considered the best choice at that time. The students are perplexed further by the negative attitudes of their own seniors, teachers and the complexity of the society we live. Having chosen medicine as a profession, the best way to deal with it is to go all out and not be contented with half hearted efforts at it. Happiness in life lies in doing something closest to our souls in the best possible manner. The following paragraph from one of the most respected physicians of our time emphasizes the scope of medicine in all its dimensions.

No greater opportunity or obligation can fall to the lot of a human being than to become a physician. In the care of the suffering he needs technical skill, scientific knowledge, and human understanding. He who uses these with courage, with humility, and with wisdom will provide a unique service to his fellow man, and will build an enduring edifice of character within himself. The physician should ask of his destiny no more than this; he should be content with no less.

Tinsely R. Harrison

The limits to medicine are best expressed by Trudeau:

To cure sometimes, to relieve often, to comfort always.

To be able to do this we require to cultivate attitudes that enable us to gain a broad knowledge of the subject of medicine, technical skills and the wisdom to use them well. In the dedication to his volume *Underwoods*, Stevenson wrote:

There are men and classes of men that stand above the common herd: the soldier, the sailor, and the shepherd not infrequently; the artist rarely; rarer still, the clergyman; the physician almost as a rule. He is the flower (such as it is) of our civilization; and that stage of man is done with, and only remembered to be marveled at in history, he will be thought to have shared as little as any in the defects of the period, and most notably exhibited the virtues of the race. Generosity he has, such as is possible to those who practice an art, never to those who drive a trade; discretion, tested by a hundred secrets; tact, tried in a thousand embarrassments; and what are more important, Herculean cheerfulness and courage. So it is that which brings air and cheer into the sick-room, and often enough, though not as often as he wishes, brings healing.

William Osler maintained that “The practice of medicine is an art, not a trade; a calling, not a business; a calling in which your heart will be exercised equally with your head...”

The science of medicine is easily learned in our training. To practice medicine as an art requires life long commitment and dedication. The practice of any art requires style. As we practice the art of medicine, develop a style or a manner of our own, a method by which we deal with our patients, students and colleagues. The ingredients of an ideal physician involve compassion, gentleness toward patients, a balanced disposition and coolness of mind especially under stress. The ability to communicate well is particularly important as most of what we do involves either teaching patients or students.

In his address to the medical students at Harvard Medical School in 1927, Dr. Peabody said:

The practice of medicine in its broadest sense includes the whole relationship of the physician with the patient. It is an art based to an increasing extent on the medical sciences but comprising much that still remains outside the realm of any science. The art of medicine and the science of medicine are not antagonistic but supplementary to each other. There is no more contradiction between the science of medicine and the art of medicine than between the science of aeronautics and the art of flying. Good practice presupposes an understanding of the sciences that contribute to the structure of modern medicine, but it is obvious that sound professional training should include a much broader equipment.

The treatment of disease may be entirely impersonal; the care of a patient must be completely personal. The significance of the intimate personal relationship between physician and patient cannot be too strongly emphasized, for in an extraordinarily large number of cases both diagnosis and treatment are directly dependent on it, and failure of the young physician to establish this relationship accounts for much of his ineffectiveness in the care of patients. What is spoken of as a clinical picture is not just a photograph of a man sick in bed; it is an impressionistic painting of the patient surrounded by his home, his work, his relationship, his friends, his joys, sorrows, hopes, and fears. Thus the physician, who attempts to take care of patient while he neglects those factors

THE SCIENCE AND ART OF MEDICINE

that contribute to the emotional life of his patient, is as unscientific as the investigator who neglects to control all the conditions that may affect his experiment. The good physician knows his patients through and through, and his knowledge is bought dearly. Time, sympathy, and understanding must be lavishly dispensed, but the reward is to be found in that personal bond which forms the greatest satisfaction of the practice of medicine. One of the essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient.

His instruction is even more pertinent today than in 1927. The requisites of a profession are best summarized by Tuttle and are particularly applicable to medicine.

The professional man is in essence one who provides service. But the service he renders is something more than that of a laborer, even the skilled laborer. It is a service that wells up from the entire complex of his personality. True, some specialized and highly developed techniques may be included, but their mode of expression is given its deepest meaning by the personality of the practitioner. In a very real sense his professional service cannot be separated from his personal being. He has no goods to sell, no right price for service, for what is the share of a man's worth? If he does not contain the quality of integrity, he is worthless. If he does, he is priceless. The value is either nothing or it is infinite. So do not set a price on yourself. Do not measure out your professional services on an apothecary's scale and say only this for so much. Do not debase yourself by equating your souls to what they will bring in the market. Do not be a miser, hoarding your talent and abilities and knowledge either among yourself or in dealing with your clients, patients, flock. Rather be reckless and spendthrift, pouring out your talent to all to whom it can be of service. Throw it away, waste it, and in the spending it can be of service. Do not keep a watchful eye lest you slip and give away a little bit of what you might have sold. Do not censor your thoughts to gain a wider audience. Like love, talent is useful only in its expenditure, and it is never exhausted. Certain it is that one must eat, so set what price you must, on your service. But never confuse the performance, which is great, with the compensation, be it money, power, or fame, which is trivial.

All this may be considered too idealistic and impractical by present day standards and requirements. Everybody is equally concerned about the state of affairs in medicine today. The best way to deal with this problem appears to be to take care of one's own attitudes first. One should avoid being judgmental in dealing with others but show that right things are still possible, by personal example. Most of us start as ideal medical students, progress to near ideal postgraduates, good assistant professors, and finally metamorphose in to that ultimate achievement, the terrible professor. To maintain these ideals one must constantly struggle. Systems within which we operate are far from perfect and the personal influence of the teacher can to some extent compensate for the inadequate academic system. Cultivate the following qualities and propagate them. Some of the qualities are elaborated but others are self explanatory.

CLINICAL METHODS IN CARDIOLOGY

<i>Qualities of a physician</i>
Enthusiasm A full personal knowledge of the branch practised/taught A sense of obligation to teach The art of detachment A systematic method Thoroughness Honesty Attitude Appearance Humility Unreserved respect for excellence The conviction that right things are possible Certain degree of insensitivity or obtuseness to criticism Willingness to take another opinion in the best interest of the patient

1. ***Enthusiasm:*** One should have deep love for the subject and people. The desire to teach and care for people, without which all medical knowledge becomes cold and lifeless. Do not take up a subject that doesn't interest you for any length of time. By doing this, you are not only harming yourself but also the patients under your care.

2. ***A full personal knowledge of the branch taught:*** Not second hand information derived from books, but the living experience derived from practical, well tested experience of a lifetime.

3. ***A sense of obligation:*** The feeling which impels a teacher to also be a contributor, and to add to the stores of medical knowledge from which we so freely draw to teach and practice medicine.

4. ***Art of detachment:*** The faculty of isolating ourselves from the pursuits and pleasures incident to routine life and an emotional detachment to the diagnoses we make. In all matters medical, what is right is more important than who is right.

5. ***Systematic approach:*** Unless one is a genius, a systematic method is essential to learn medicine. We must plan each day of ours in such a way that minimum time is wasted in unnecessary things. What we do daily is going to decide what we are going to be at the end of a year or two. These few years as a student are going to make or break your career. The present system of medical education does not foster competence and conviction in the student or future doctor. By the time the

students finish their course, they are hardly in a position to take care of patients because they have not spent enough time in the hospital. On many days in a week, the student neither examines nor even talks to any patient. During their clinical years, students are not given any clinical responsibilities. In order to learn to deal with patients they should spend their time taking care of patients as house physicians or residents do. In the present system, it is not rare that by the time they finish their medical course, many find it difficult to communicate with patients.

6. *Thoroughness*: It is essential in all medical matters, be it a preparation for a talk, examination, or patient evaluation and management.

7. *Honesty*: The ability to admit a mistake, take another opinion or help when we are not sure requires courage and conviction on our part. We must conduct ourselves in an irreproachable manner so that not even the slightest doubt would be raised about our integrity.

8. *Attitude*: A doctor should be tolerant and patient. We should avoid judging people and taking sides because we undertake to take care of everybody irrespective of their origin or status.

9. *Appearance*: We must pay attention to appearance and behaviour as society often tends to judge us on this basis. A dignified and cheerful manner is particularly important in dealing with sick people.

10. *The grace of humility*: Whatever excellence one achieves in medicine, there can never be perfection in it. There are always places to go and people to meet from whom we can learn to do better things. This realization makes us humble and without this quality, one stands out as an intolerable character.

11. *Unreserved respect for excellence*: Excellence in any branch of science or medicine, from whatever person, institution or country it emanates, should be respected and duly acknowledged. It is true that healthy competition or rivalry helps in achieving the higher objectives in medicine, but when carried too far, it becomes counter-productive.

12. *Unswerving conviction that good things are possible*: In the present atmosphere of medical practice and medical education, contributed to by the profession, the politician, the bureaucrat, it is easy to give up all hope of doing anything extraordinary and to become part of the corrupt system. Only the strong

conviction that right things are still possible and the courage to withstand the pressures and put up with criticism helps to achieve the desired goals. These ideals should be maintained in spite of heavy odds in one's day to day work. The best time to start learning these attitudes is now when one is a student although it is never too late even for an older doctor. The attitude towards friends, classmates, seniors, juniors, patients and their families is an indicator of what one is going to be. This is the time one must learn to interact with people and patients. It is not enough to be a good student. One must strive to be a likable person in the college, hospital, and home. Once cultivated, these habits like bad habits, are contagious. The best target is the students at various levels who are yet to be spoiled by exposure to the tricks of the trade of medicine today.

While examining and evaluating patients

Tact, sympathy and understanding are expected of the physician, for the patient is no mere collection of symptoms, signs, disordered functions, damaged organs, and disturbed emotions. He is human, fearful and hopeful, seeking relief, help and reassurance. To the physician as to the anthropologist, nothing human is strange or repulsive. The misanthrope may become a smart diagnostician of organic disease. But he can scarcely hope to succeed as a physician. The true physician has a Shakespearean breadth of interest in the wise and the foolish, the proud and the humble, the stoic hero and the whining rogue. He cares for people.

Tinsely R. Harrison

While we do our best we should be prepared to face thanklessness and even exploitation from some people. A certain degree of obtuseness or insensitivity to criticism is sometimes necessary in a professional.

Finally, all that is done in medicine is based on the conviction, that human life is valuable and human beings require to be treated with dignity and respect. As a doctor if your first reaction to the person is suspicion and hatred, one should not pursue a career in clinical medicine. Those of us who are religiously oriented should realize that our religion is medicine and all other religions pale before it.

A good example is the best sermon.

(Anonymous)

With the rapid advances that occurred in the last decade, medicine has become more remunerative and extremely competitive. The competitive atmosphere brought out the best in some individuals and institutions leading to superlative performance. However some institutions and individuals wilted morally under

THE SCIENCE AND ART OF MEDICINE

this pressure and took recourse to devious methods of dealing with the problem. For them, each patient is a prospect on whom almost all tests and few procedures can be done. They later call up the referring doctor and tell him or her how much money can be made on that patient. Unnecessary investigations, surgeries, or interventions have become commonplace. However the system of fee-splitting is destroying the soul of medical practice. This practice takes away all the trust that patients come to us with. This distrust will continue to grow if medicine is debased with such practices.

The tragedy of life is what dies inside a man while he lives.

2 Patient Presentations

Nothing clarifies the issue better than stating the case to someone else.

Sherlock Holmes

The way one presents a patient's case is an expression of the way of thinking and perception of the patient's problem. Presenting the patient's case is an art that can be described only partly but can be learned with practice.

OUTLINE OF PRESENTATION

There are two varieties of presentations:

1. Short or brief presentations,
2. A complete 7–10 minute presentation.

A good presentation requires sufficient knowledge of the patient and writing up the patient details. The index card or a note book are best suited for this purpose. In the initial stage of your career a number of facts may have to be written, but as your experience increases, you need to write less.

Note the following features (on index card)

1. Patient information : Address, referring doctor with phone number
2. The introductory : sentence
3. Chief complaint : Note salient points and duration
4. History of : Relevant negative and positive history, family history, risks present illness

PATIENT PRESENTATIONS

5. Past medical history : Note active problems (PMH)
6. Allergies : For drugs, foods, type of reaction
7. Drug therapy : Past, present and response. Alcohol, tobacco
8. Review of systems : Note any positive history (ROS)
9. Physical exam (PE) : Introductory sentence, vital signs. Only positive findings
10. Lab tests : Positive findings
11. Problem list :

OUTLINE OF BRIEF PRESENTATION

The brief presentation is a 2–3 minute summary for the service round in the morning. It is intended to orient the members of the team who do not know the patient at all (or chiefs who come for the round only to have some fun).

1. Introductory :
sentence
2. Chief complaint :
3. History of : In a condensed form, summarize the history of the present illness
present illness and duration
4. Drugs :
5. Physical : General condition, good, stable, unstable, drowsy, comatose
examination
Vital signs : Stable, or those that are abnormal
6. Relevant lab : Positive findings
tests
7. Summary : Brief
8. Plan : Of what is to be done next. For example, waiting for discharge or angiogram.

General principles of brief presentation

1. This is intended to apprise the members of the team and elicit their opinion of the patient.
2. Any additional information can be provided if asked for.
3. Speak clearly and decisively.

4. Bring out the active problems and also what investigation is awaited. This is helpful in taking over patients and to cover patients at night. This ensures continuity of care.

Brief presentation (sample)

This is Mrs. R.S.'s case and is the 4th day of hospitalization. She is a 50-year-old Hindi speaking housewife with a history of uncontrolled hypertension and diabetes. She was hospitalized with prolonged chest discomfort typical of acute myocardial infarction more than 12 hours after the onset of pain. She was taking Hydrochlorthiazide 25 mg per day and Enalapril 15 mg twice a day. On physical examination, she is stable with stable vital signs. Pulse: 64/min, regular; respiratory rate: 20/min; BP: 130/90 mmHg; JVP: normal, no cardiac enlargement, LV S4 was heard. Lungs: clear; other systems: Normal.

Lab tests: ECGs showed serial changes of acute inferior MI

Today's ECG: Normal sinus rhythm, 64/min regular, P-R = 0.20 sec and features of transmural inferior MI without RV infarction lateral extension or true posterior MI. Other lab tests are normal. X-ray chest is normal.

Presently she is on isosorbide dinitrate 10 mg three times a day, aspirin 325 mg/day and diazepam 5 mg twice a day.

Plan: She is waiting to complete the hospital course of 1 week and have an echocardiogram and functional testing prior to discharge.

FORMAL DETAILED PRESENTATION

1. Patient information: 48-year-old business-man was perfectly well before 14 April etc.
2. Introductory sentence:
3. Chief complaints and duration: In the presentation, unlike write up, avoid using the patient's words. Give the description that focuses quickly on the problem at hand.
4. History of present illness: Present a succinct version of the history of present illness. Give positive findings from review of systems section. Mention risk factors and family history.
5. Past medical history: Prior admissions, other active medical problems, investigations done in the past and their results.

PATIENT PRESENTATIONS

6. Allergies: Drug reactions
7. Medications: All present drugs with their dosage and response to them.
8. Review of other systems: Positive features, pertinent positive symptoms other than those mentioned during history of present illness.
9. Physical examination: Introductory sentence describing appearance and condition. (e.g.: patient comfortable, toxic or in respiratory distress, etc.)
Vital signs
Pertinent positive findings
10. Lab tests: Pertinent positive and negative reports if they are significant.
11. Summary: Give a brief 2–3 sentence summary and then pause so that the discussion is initiated by the consultant.

Case presentation guidelines

1. The routine round is initiated by the case presentation of the previous night's admissions. The following aspects are usually discussed.
 - Symptoms of the patient, their interpretation, conclusions drawn
 - Physical signs, their pathophysiology
 - Differential diagnosis
 - Pathophysiologic mechanisms
 - Complications of the disease
 - Tests to ask for, their interpretation, sensitivity, specificity
 - Drug therapy: Indications, contraindications, side effects, findings from major trials completed and ongoing
 - Future plan
 - Natural history of the disease diagnosed
 - Indications and results of interventions, findings from major studies.
2. You must feel confident and look confident. When you present a patient with mitral stenosis, even if others in the room know more about mitral stenosis, your patients's mitral stenosis is better known to you.
3. Speed of presentation: Relax and speak as though you are talking to your friends (on a serious issue).
4. Do not read your presentation.
5. Speak precisely: Do not say the blood pressure is around 120/80 mmHg. Say the blood pressure is 120/80 mmHg.

6. Speak briefly: The brief presentation is meant to be brief. Even the formal presentations should be succinct.
7. Omissions/condensations: Describe only the positive and negative findings relevant to the patient at hand. Purposefully omit the details that are not relevant to the 'argument' you are putting forward.
8. Physical findings: It is common at all levels of clinical experience to have equivocal physical findings. Present your findings 'if others disagreed, simply say, another observer thought differently'.
9. At the end of the history, physical exam and lab data presentations, summarize the case in two or three sentences. For example, in summary, Mrs. M 58-year-old with history of hypertension, diabetes and hyperlipidemia, presented with acute inferior infarction of 4 hours duration and has no contraindication to thrombolytic therapy.
10. Hospital course: For patients with multiple active problems describe the hospital course in a problem oriented manner.
11. When you are proven wrong by the bedside : Patients often change histories. This happens frequently to all of us and one should not feel inadequate or annoyed about it. Consider the implications of the new information made available.
12. The patient is the centre of importance: It is a good practice to have the presentations away from the patient's bedside. If the patient interrupts the discussion by some questions or information answer those first. Do not use words like death, severe, irreversible etc., in front of the patient. They are likely to be misinterpreted and misunderstood.
13. In all matters medical, what is right is more important than who is right. *Do not get emotionally attached to the diagnosis you make.*

In order to learn from the experience of an individual patient, you must read about each of them while they are under your care. Reading about each patient involves reading and discussing about their symptoms, signs, diagnosis, differential diagnosis, laboratory tests, the drugs and any interventions when applicable. This should be done by the end of the day.

3 Cardiovascular Diagnosis

During ward rounds and bedside discussions one may sometimes come across the resident doctor giving an incomplete diagnosis after a painstaking description of the history and physical findings. This is a disappointing and annoying sight for the teacher.

A comprehensive cardiovascular diagnosis is a prerequisite for appropriate decision-making in patients with heart disease.

<i>Components of cardiac diagnosis</i>
Etiologic Anatomic defect with severity Functional derangement with severity Rhythm and rate Associated lesions Complications Functional categorization Specific recommendations Diagnostic Therapeutic

For example, in a patient with valvular disease (mitral stenosis) the expression should be:

- Chronic rheumatic heart disease
- Mitral stenosis (severe) calcific, severe subvalvular fusion
- Pulmonary arterial hypertension (severe)
- Right ventricular failure (severe)
- Tricuspid regurgitation functional (severe)
- Atrial fibrillation (ventricular rate = 120/min)
- NYHA functional class IV

- No rheumatic activity
- No infective endocarditis.

The importance of each of these features can not be overemphasized. Mild mitral stenosis does not require any intervention but moderate or tight mitral stenosis needs surgery. A calcified valve/subvalvular fusion makes the valve unsuitable for closed mitral valvotomy (CMV), and an open valvotomy (OMV) may be suitable for milder subvalvular fusion. Mitral valve replacement (MVR) may be the only choice when the valve is calcific with subvalvular fusion. As the severity of pulmonary arterial hypertension (PAH) increases, the risk of surgery also increases.

Right ventricular failure (RVF) and tricuspid regurgitation (TR) have similar implication and require control with digitalis and diuretics before surgery. Atrial fibrillation (AF) in a young person with mitral stenosis usually means a tight and longstanding lesion. It is important to make a note of the ventricular rate in atrial fibrillation as rapid rates aggravate pulmonary venous hypertension (PVH) and precipitate pulmonary edema by reduction in diastolic filling time. Digitalis is the drug of choice to reduce the ventricular rate. Due to the risk of peripheral embolism, anticoagulants are indicated in atrial fibrillation. The risk of surgery is very high in patients with class IV (NYHA) and is low with class II and III.

CONGENITAL HEART DISEASE

Broadly two categories exist: acyanotic and cyanotic. For example, when a diagnosis of atrial septal defect is made, the diagnostic expression should be:

Congenital acyanotic heart disease
 Left to right shunt
 Atrial septal defect (ostium secundum)
 Pulmonary to systemic flow ratio of $> 2:1$
 Pulmonary arterial hypertension (mild) hyperkinetic
 No congestive cardiac failure
 Normal sinus rhythm
 Class II (NYHA), no associated defects.

In left to right shunts, the following features should be noted; these are particularly applicable to ventricular septal defects.

- a. Site of the defect

CARDIOVASCULAR DIAGNOSIS

- b. Size of the defect
 - c. Number of defects
 - d. Shunt pattern : left to right, balanced or right to left
 - e. Pulmonary to systemic flow ratio ($> 2:1$ or $< 2:1$)
 - f. Pulmonary arterial hypertension. presence/absence and severity. Flow dependent (hyperkinetic) or resistance dependent (fixed)
 - g. Congestive cardiac failure
 - h. Associated defects
 - i. Isolated or part of a complex defect
 - j. Presence or absence of infective endocarditis, and
 - k. Height, weight, head circumference and growth percentile.
- The NYHA functional categorization is not applicable in children.

CYANOTIC HEART DISEASE

Two forms exist:

increased pulmonary flow, and reduced pulmonary flow. Normal pulmonary flow is not possible with any cyanotic heart disease.

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Example:

Congenital cyanotic heart disease,
Diminished pulmonary blood flow,
Tetralogy of Fallot (mild/moderate/severe),
No congestive cardiac failure,
Normal sinus rhythm,
Height, weight, head circumference, growth percentile
Presence or absence of spells, brain abscess, infective endocarditis.

Any increased pulmonary blood flow in cyanotic heart disease, indicates some form of transposition of great arteries or pulmonary veins (D-TGA or TAPVC) or an admixture lesion like single ventricle or double outlet right ventricle.

Example:

Congenital cyanotic heart disease,
Increased pulmonary blood flow,
D-transposition of great arteries,
Ventricular septal defect (large),
Congestive cardiac failure,

Pulmonary arterial hypertension (hyperkinetic),
Height, weight, head circumference, growth percentile.

CORONARY ARTERY DISEASE

The common clinical syndromes are:

- Chronic stable angina
- Acute myocardial infarction
- Unstable angina.

Example:

- Coronary artery disease
- Acute anterior myocardial infarction of 3 hours duration
- Past chronic stable angina of 2 years duration
- No past myocardial infarction
- Normal sinus rhythm, ventricular ectopics < 6/min
- Hemodynamic subset: left ventricular failure mild
- No evidence of acid peptic disease or contraindication to thrombolytic therapy

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Time from the onset of pain in acute myocardial infarction is important as interventions to prevent myocardial necrosis are most useful in the first 6 hours. Previous history of chronic stable angina is indicative of obstructive coronary disease progressing to acute myocardial infarction and coronary spasm is unlikely. In the initial evaluation of acute myocardial infarction, spasm needs to be distinguished from obstructive coronary disease. Rhythm is particularly important, as most of the deaths in the first few hours of myocardial infarction are due to arrhythmias. Hemodynamic categorization is essential to intelligent management of all patients with acute myocardial infarction.

In chronic stable angina the diagnosis should include:

- Coronary artery disease
- Angiographic right coronary 75% lesion, concentric, short segment
- Normal left ventricle function/no regional wall motion abnormality
- Chronic stable angina; functional Class III (2 years); no recent change
- Mixed angina
- No congestive cardiac failure
- Normal sinus rhythm 100/min
- No previous myocardial infarction

CARDIOVASCULAR DIAGNOSIS

Risks: Hypertension mild, and

Plan: Medical therapy/Suggest angioplasty.

Duration of angina: When it is of more than one year duration the coronary lesion is usually non-calcific and responds well to percutaneous transluminal

Table 3.1: Hemodynamic subsets of AMI

Subset	PA (mean) mmHg	PAW/mmHg	CI/l/min/m ²	Action	Remarks
1. Normal	< 15	< 12	2.5–3.5	None	
2. Hyperdynamic	< 15	< 12	3.0	β-blockers	Tachycardia, HIN
3. Hypotension + hypovolemia	< 15	< 9	< 2.7	Volume load	Reclassify after PAW is 14–18 mmHg
4. LVF, mild	> 22	18–22	< 2.5	Diuretic Vasodilator	Dyspnea, Lung rales
5. LVF, severe	> 25	> 22	< 1.8	IABP PPV	Lung edema
6. Cardiogenic shock	> 22	> 18	< 1.8	IABP Dopamine Dobutamine Emergent PTCA/CABGS	

PPV = Positive pressure ventilator,

IABP = Intraaortic balloon pump.

Table 3.2: Mortality in AMI

Based on clinical examination			Based on invasive monitoring		
Class	Definition	GUSTO-I Mortality (%)	Subset	Definition	Mortality (%)
I	Rales and S ₃ absent	5.1	I	Normal hemodynamics PCWP < 18. CI > 2.2	2
II	Rales over < 50% of lung	13.6	II	Pulmonary congestion PCWP > 18. CI < 2.2	10
III	Rales over > 50% of lung fields (pulmonary edema)	32.2	III	Peripheral hypoperfusion PCWP < 18. CI > 2.2	22
IV	Shock	57.8	IV	Pulmonary congestion and Peripheral hypoperfusion PCWP > 18. CI < 2.2	56

coronary angioplasty (PTCA). Beyond an year, calcific lesions with diffuse and multivessel disease are more likely and are unsuitable for PTCA. With the exception of left main disease, Canadian Cardiovascular Society class I and II patients do well with medical therapy and do not need surgery whereas class III and IV patients are benefited by coronary bypass graft surgery. The classic exertional angina or fixed threshold angina is due to obstructive coronary disease and responds to betablockers. In variable threshold angina, dynamic obstruction caused by vasoconstriction plays an important role in causing myocardial ischemia. The person may be capable of substantial physical activity at one time, while at another time minimal activity results in angina. Most of these patients have an underlying fixed obstruction with superimposed spasm. The term mixed angina is applied to this group of patients and they are best treated by calcium blockers and betablockers. Betablockers alone may aggravate spasm. Heart rate has obvious implication in selecting drug therapy.

PRIMARY MYOCARDIAL DISEASE

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Is the structural and functional abnormality of heart muscle unrelated to

- Valvular
- Coronary
- Hypertensive
- Pericardial and pulmonary disease?

If the answer is yes, a diagnosis of cardiomyopathy is made. Once the diagnosis of cardiomyopathy is made, the following questions should be asked.

1. Is it acute or chronic?
2. Is it focal or diffuse?
3. Which clinical syndrome?
 - a. Congestive (dilated)
 - b. Restrictive
 - c. Obliterative
 - d. Hypertrophy: obstructive or non-obstructive
4. Is there myocardial failure?
5. Is there AV valve regurgitation?
6. Is there an arrhythmia?
7. Are there any identifiable etiologic factors?

8. Functional categorization (NYHA)

Examples:

Cardiomyopathy, chronic diffuse congestive

Biventricular failure

Mitral regurgitation (functional)

Paroxysmal atrial tachycardia

Chronic alcoholism

Class III NYHA

Classification of cardiomyopathies

Specific cardiomyopathies

- Ischemic cardiomyopathy
- Valvular cardiomyopathy
- Hypertensive cardiomyopathy
- Inflammatory cardiomyopathy

Myocarditis

Idiopathic

Autoimmune

Infectious

Chagas disease

HIV

Enterovirus

Adenovirus

Cytomegalovirus

Bacterial (endocarditis/myocarditis)

Metabolic

- Endocrine
 - Thyrotoxicosis
 - Hypothyroidism
 - Adrenal cortical insufficiency
 - Pheochromocytoma
 - Acromegaly
 - Diabetes mellitus

- Familial storage disease/infiltration
 - Hemochromatosis
 - Glycogen storage disease
 - Hurler's syndrome
 - Refsum's syndrome
- Neimann-Pick disease
 - Hand-Schuler-Christian disease
 - Febry-Anderson disease
 - Morquito-Ullrich disease

Dilated cardiomyopathy
Hypertrophic cardiomyopathy
Restrictive cardiomyopathy
Arrhythmogenic right ventricularcardiodysplasia

Unclassified cardiomyopathies

- Atypical presentation
 - Fibroelastosis
 - Non-compacted myocardium
 - Systolic dysfunction without dilation
 - Mitochondrial cardiomyopathy
- Mixed presentation
 - Amyloidosis
 - Hypertension

4 Functional Categorization of Cardiovascular Disability

Most diseases have an effect on the functional capacity of the patient. In cardiovascular disease, the severity of the functional loss is also an important marker of the prognosis. Often this decides the therapeutic choice. Three methods are presently used to classify the severity of cardiovascular disease in terms of functional capacity.

- New York Heart Association functional classification (NYHA)
- Canadian Cardiovascular Society functional classification (CCS)
- Specific Activity Scale of Goldman (SAS)

The NYHA classification is widely used and is applicable to any symptom that is disabling. The CCS classification is applicable only to angina pectoris and is currently used in all clinical trials on coronary artery disease. The SAS classification is more objective but is used less often.

NYHA FUNCTIONAL CLASSIFICATION

Class 1: Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea or anginal pain.

Class 2: Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea or anginal pain.

Class 3: Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes

fatigue, palpitation, dyspnea, or anginal pain.

Class 4: Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort increases.

CCS FUNCTIONAL CLASSIFICATION FOR ANGINA

Class 1: Ordinary physical activity, such as walking and climbing stairs, does not cause angina. Angina with strenuous or rapid or prolonged exertion at work or recreation.

Class 2: Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold weather, in wind, or when under emotional stress, or only during the few hours after waking, walking more than two blocks on level ground and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions.

Class 3: Marked limitation of ordinary physical activity. Walking one or two blocks on level ground and climbing more than one flight in ordinary conditions.

Class 4: Inability to carry on any physical activity without discomfort. Anginal syndrome may be present at rest.

SPECIFIC ACTIVITY SCALE (SAS)

The intensity of physical activity can be expressed in metabolic units (MET or metabolic equivalents of oxygen consumption). The metabolic unit is the ratio of metabolic rate during exercise to metabolic rate at rest. One metabolic unit corresponds to energy expenditure of approximately 1 kcal per kg of body weight per hour, or an oxygen uptake of 3.5 ml per kg per minute.

Class 1: Patients can perform to completion any activity requiring 7 metabolic equivalents, for example, he can carry 24 lb up eight steps, carry objects that weigh 80 lb (35 kg), outdoor work like spade soil, recreational activities (jog/walk 5 mph or about 8 kmph)

Class 2: Patients can perform to completion any activity requiring 5 metabolic equivalents but cannot and do not perform to completion activities requiring >7 metabolic equivalents, for example, have sexual intercourse without stopping, garden or walk at 4 mph (6 kmph) on level ground.

Class 3: Patients can perform to completion any activity requiring 2 metabolic equivalents but cannot and do not perform to completion any activities requiring >5 metabolic equivalents, for example, shower without stopping, strip and dress without stopping, walk 2.5 mph (4 kmph)

Class 4: Patients cannot and do not perform to completion activities requiring >2 metabolic equivalents, cannot carry out activities listed under class 3.

Whatever method is employed, it is good practice to specify the functional class with therapy or without therapy. Further it should be understood that this method of evaluation is best suited for certain types of heart disease only and in situations like arrhythmias, the functional classifications become irrelevant. Also certain dynamic pathophysiological conditions and non-cardiac illnesses like anemia may suddenly change the functional class of the patient.

NATURE OF WORK

Sedentary work: Sedentary work is defined as ‘lifting maximum 10 lb and occasionally carrying such articles as books, ledgers, and small tools. Although a sedentary job usually involves sitting, a certain amount of walking and standing may be necessary in carrying out duties associated with the job. Jobs are sedentary if walking and standing are required only occasionally and other sedentary criteria are met’.

Light work: Light work involves ‘lifting maximum 20 lb with frequent lifting and carrying of objects weighing up to 10 lb. Even though the weight lifted may be only a negligible amount, a job is included in this category when it requires walking or standing to a significant degree, or when it involves sitting most of the time with a degree of pushing and pulling of arm and or leg controls’.

Medium work: This includes ‘lifting 50 lb maximum with frequent lifting and/or carrying of objects weighing up to 50 lb’.

Heavy work: This is defined as ‘lifting 100 lb maximum with frequent lifting and/or carrying of objects weighing up to 50 lb’.

Very heavy work: This involves ‘lifting objects in excess of 100 lb with frequent lifting and/or carrying of objects weighing 50 lb or more’.

Both men and women who did more than 2 hours of conditioning physical activity a week and men with a maximal oxygen uptake of at least 2.7 litres per minute, or 34 ml per kg per minute (10 METS exercise capacity) had less than half the risk of acute myocardial infarction of the least active or the least fit men.

Table 4.1: Assessing cardiovascular disability

<i>Canadian Cardiovascular Society functional classification</i>	<i>Specific Activity Scale</i>
Ordinary physical activity, such as walking and climbing stairs does not cause angina. Angina with strenuous or rapid or prolonged exertion at work or recreation.	Patients can perform to completion any activity requiring ≤ 7 metabolic equivalents, e.g. can carry 24 lb up eight steps; carry objects that weigh 80 lb; do outdoor work (shovel snow, spade soil); and do recreational activities (skiing, basketball, squash, handball, jog/walk 5 mph).
Slight limitation of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, in wind or when under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions.	Patients can perform to completion any activity requiring ≤ 5 metabolic equivalents, e.g., having sexual intercourse without stopping, roller-skate, rake, dance, fox trot, walk at 4 miles per hour on level ground, but cannot and do not perform to completion activities requiring ≤ 7 metabolic equivalents.
Marked limitation of ordinary physical activity. Walking blocks on one to two the level and climbing more than one flight in normal conditions.	Patients can perform to completion any activity requiring ≤ 2 metabolic equivalents, e.g., shower without stopping, strip and make bed, clean windows, walk 2.5 mph, bowl, play golf, dress without stopping, but cannot and do not perform to completion any activities requiring ≤ 5 metabolic equivalents.
Inability to perform any physical activity without discomfort. Anginal syndrome may be present at rest	Patients cannot or do not perform to completion activities requiring ≤ 2 metabolic equivalents.

Adapted from Goldman L, Cook EF, Loscalzo A. Comparative reproducibility and validity of systems for assessing cardiovascular functional class: Advantages of a new specific activity scale. *Circulation* 1981; 64:1227.

FUNCTIONAL CATEGORIZATION OF CARDIOVASCULAR DISABILITY

Table 4.2: NYHA functional classification

<i>Class</i>	<i>New York Heart Association functional classification</i>
I	Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, dyspnea, or anginal pain.
II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
III	Patients with cardiac disease resulting in marked limitation of physical activities. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.
IV	Patients with cardiac disease resulting in inability to perform any physical activity without discomfort.

5 Cardiovascular Dynamics: Basic Considerations

It is essential to know intracardiac pressures and flows for a proper understanding of cardiovascular disease. Often, the student feels that the knowledge of cardiac hemodynamics is unnecessary for a student and is only meant for specialists. It must be realized that this is a prerequisite for a proper understanding of the patient's symptoms or signs and the results of any laboratory test.

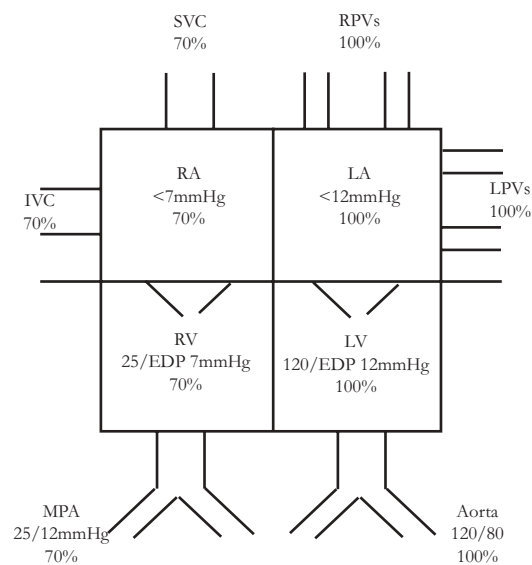


Fig. 5.1: Depiction of normal intracardiac pressures and oxygen saturation in various chambers

Normal aortic pressure is 120/80 mmHg. The left ventricular peak systolic pressure and aortic systolic pressure are equal as there is no pressure difference across a normal aortic valve and left ventricle; it behaves as a common chamber in systole. The left ventricular end-diastolic pressure is normally < 12 mmHg. In diastole when the mitral valve is open, the left ventricle and atrium behave like a common chamber, and thus the left atrial mean pressure does not normally exceed 12 mmHg. Pulmonary capillary wedge pressure (< 12 mmHg) reflects the pressure in pulmonary veins, which are in continuity with left atrium. In the absence of pulmonary vascular disease, the pulmonary artery diastolic pressure is equivalent to the capillary wedge pressure. Thus all these five pressures – left ventricular diastolic, left atrial mean, pulmonary venous, pulmonary capillary wedge and pulmonary artery diastolic – are equal in the absence of disease. Thus,

LV diastolic pressure = LA mean pressure = Pulmonary venous pressure = Pulmonary capillary wedge pressure = Pulmonary artery diastolic pressure = 12 mmHg

Consequently, in routine practice the measurement of pulmonary artery pressure will give information about the left heart filling pressures. Similarly on the right side, the peak right ventricular pressure is equal to pulmonary artery systolic pressure, and the right ventricular diastolic pressure is equal to mean right atrial pressure and the jugular venous pressure. Normal values for hemodynamic parameters are given in Table 5.1 and Table 5.2.

The oxygen saturation of blood returning from tissues is around 70 per cent as 5 ml of oxygen per 100 ml blood is normally extracted in the tissues. After oxygenation in the lungs, the saturation becomes 100 per cent in the left sided chambers starting with pulmonary veins. As bronchial veins drain into pulmonary veins, and myocardial veins or *thebesian veins* drain directly into the left ventricle, saturation in the left ventricle may not always be 100 per cent but is usually above 95 per cent. Step-up or step-down at any chamber level is indicative of either left to right or right to left shunt.

CARDIAC OUTPUT

Normal cardiac function is the ability to pump adequate blood, commensurate with the tissue need, even at maximal exercise without elevation of filling pressures.

Table 5.1: Normal values for hemodynamic parameters and wave patterns

	<i>Pressures (mmHg)</i>
<i>Systemic</i>	
Peak systolic	100–140
End-diastolic	60–90
Mean (Diastolic + 1/3 of pulse pressure)	
<i>Left ventricle</i>	
Peak systolic	100–140
End-diastolic	3–12
<i>Left atrium or pulmonary wedge</i>	
Mean	3–10
a wave	3–12
v wave	3–12
<i>Pulmonary artery</i>	
Peak systolic	15–30
End diastolic	4–12
Mean	9–18
<i>Right ventricle</i>	
Peak systolic	15–30
End diastolic	4–12
<i>Right atrium</i>	
Mean	2–7
a wave	3–10
v wave	3–10

Table 5.2: Cardiac output and vascular resistance

Cardiac output	4–8 l/min
Cardiac index	2.6–4.2 l/min
Systemic vascular resistance	700–1600 dynes/sec/cm ⁵
Pulmonary vascular resistance	20–120 dynes/sec/cm ⁵
Oxygen consumption	110–150 ml/min/m ²
Arteriovenous oxygen difference	30–50 ml/l

The cardiac output is determined by four major factors: preload, afterload, contractility and heart rate (Table 5.3).

At any measure of contractility, the extent of muscle fibre shortening varies directly with preload, and inversely with afterload. The preload is the venous return

and the afterload is the systemic vascular resistance or aortic pressure. Apart from systemic vascular resistance, afterload is influenced by all the factors that constitute aortic impedance. Aortic impedance is the ratio of pressure to flow in the aorta, and is determined by the physical properties of blood and vascular wall (Table 5.4).

The preload, afterload and contractility are often altered in cardiovascular disease (Table 5.5).

There are serious limitations in using cardiac output as a measure of cardiac performance. The cardiac output may remain normal at rest even in heart failure but may fail to increase with exercise. This is because of alterations in preload in heart failure.

Ejection fraction

Ejection fraction is a better measure for evaluating systolic emptying of the heart (Table 5.6).

$$\text{Ejection fraction (EF)} = \frac{\text{End-diastolic volume} - \text{End-systolic volume}}{\text{End-diastolic volume}}$$

(Normal: 55–80%)

Table 5.3: Determinants of cardiac output

<i>Determinant</i>	<i>Definition</i>
Preload	Load the ventricle has to overcome before it begins contracting
Afterload	Load the ventricle has to overcome after it begins contracting
Contractility	Intrinsic ability of the heart muscle to contract independent of preload and afterload
Heart rate	The rate at which the heart pumps blood

Table 5.4: Factors influencing aortic impedance

Physical properties of blood	Viscosity of blood Anemia Polycythemia Density of blood
Physical properties of vascular wall	Diameter of aorta Viscoelasticity of aorta Reflected pressure and flow waves

Table 5.5: Alterations in preload, afterload and contractility

<i>Preload</i>	<i>Afterload</i>	<i>Contractility</i>
Increased in Supine/leg raising Volume infusion Renal failure Regurgitant lesions MR/AR TR/PR Left to right shunts VSD/PDA/ASD Hyperkinetic states Anemia Decreased in Hypovolemia Hemorrhage GI loss of fluid	Increased in <i>Left side</i> Systemic HTN Aortic stenosis Coarctation of aorta <i>Right side</i> PAH PS <i>Either side</i> Polycythemia Decreased in <i>Left side</i> MR Vasodilators Pregnancy AV fistula, PDA Severe anemia Fever <i>Right side</i> TR Pulmonary AV fistula Pulmonary vasodilators Oxygen Prostaglandins	Increased in Catecholamines Tachycardia Beta-2 stimulants Decreased in Myocardial ischemia/ infarction Myocarditis Cardiomyopathy Drugs (betablockers, calcium blockers, disopyramide)

Table 5.6: Ventricular volumes

LV end-diastolic volume	$72 \pm 15 \text{ ml/m}^2$
LV end-systolic volume	$20 \pm 8 \text{ ml/m}^2$
Ejection fraction	55–80%

The stroke volume, that is, volume of blood ejected in to aorta per beat (end diastolic volume – end systolic volume) may be maintained normally in heart failure by the compensatory increase in ventricular end-diastolic volume. From a normal end-diastolic volume (say 100 ml) a normal stroke volume (say 60 ml) ejected is a reflection of normal systolic performance of ventricle with a normal ejection fraction of 60 per cent. If the cardiac efficiency or ejection fraction

decreases from, say 60% to 30% stroke volume may decrease from 60 ml to 30 ml at an end-diastolic volume of 100 ml. However, the heart compensates by increasing end-diastolic volume from 100 ml to 200 ml (increase in preload). At end-diastolic volume of 200 ml, with an ejection fraction of 30% stroke volume will remain 60 ml. Thus, the heart compensates with increase in end-diastolic volume in the face of decreased efficiency.

Left ventricular ejection fraction may be characterized as follows. Normal left ventricular ejection fraction is above 56% (Mild LV dysfunction: 40–50%, Moderate LV dysfunction: 30–40%, Severe LV dysfunction: < 30%)

$$\text{Cardiac output} = \text{Heart rate} \times \text{Stroke volume}$$

(Normal: 4–8 L/min)

$$\text{Cardiac index} = \text{Cardiac output} / \text{BSA}$$

(Normal: 2.6–4.2 L/min/m²)

$$\text{Systemic vascular resistance (SVR)} = \frac{\text{Mean aortic pressure} - \text{Mean RA pressure} \times 80}{\text{Cardiac output}}$$

(Normal: 700–1600 dynes/sec/cm⁻⁵.)

$$\text{Pulmonary vascular resistance (PVR)} = \frac{\text{Mean PA pressure} - \text{Mean RA pressure} \times 80}{\text{Pulmonary blood flow (Qp)}}$$

(Normal: 20–120 dynes/sec/cm⁻⁵.)

CALCULATION OF VALVE AREA

Table 5.7: Valve areas (area in cm² and area index in cm²/m²)

<i>Normal or abnormal</i>	<i>Mitral valve</i>	<i>Aortic valve</i>	<i>Tricuspid valve</i>	<i>Pulmonary valve</i>
<i>Normal</i>	4–6	2.6–3.5	5.0–10.0	2.0–3.0
<i>Mild stenosis</i>				
Area	1.5–2.5	> 1.5	< 5.0	> 1.5
Area index		> 0.9		
<i>Moderate stenosis</i>				
Area	1.0–1.5	1.0–1.5		< 0.8
Area index		0.6–0.9		
<i>Severe stenosis</i>				
Area	< 1.0	< 1.0		< 0.5
Area index	0.4–0.6	< 0.4		

The normal function of a valve is to allow free forward flow and prevent any backward flow. When the valve becomes narrow due to any reason, the flow across the valve is directly proportional and the transvalvular pressure gradient is inversely proportional to the valve area. Gorlin and Gorlin have provided the mathematical formulas for calculating valve area.

$$\text{Mitral valve area} = \frac{\text{Diastolic mitral flow}}{38\sqrt{\text{Mean transmitral gradient}}}$$

$$\text{Aortic valve area} = \frac{\text{Systolic aortic flow}}{45\sqrt{\text{Mean transaortic gradient}}}$$

In pulmonic stenosis, valve area is not often used in actual practice. The ratio of the peak right ventricular pressure to peak systemic pressure (RV/SA) is used.

GRADING OF PS: RV/SA PRESSURE RATIO

<i>Category</i>	<i>RV/SA pressure ratio</i>	<i>Gradient (mmHg)</i>
1. Mild PS	< 0.5	< 50
2. Moderate	0.5–0.75	50–75
3. Severe	> 0.75	> 75 mmHg

VALVULAR REGURGITATIONS

$$\text{Regurgitant fraction} = \frac{\text{Angiographic stroke volume} - \text{Fick stroke volume}}{\text{Angiographic stroke volume}}$$

Table 5.8: Correlates of severity of regurgitant lesions: clinical and angiographic correlations

<i>Regurgitant fraction</i>	<i>Angiographic severity</i>	<i>Clinical severity</i>
1. < 20%	+	Trivial
2. 20–40%	++	Mild
3. 40–60%	+++	Moderate
4. > 60%	++++	Severe

The tricuspid valve area calculated by the Gorlin formula is unreliable and is not used to assess the severity of tricuspid stenosis. The gradients across the valve are often used. For a diagnosis of tricuspid stenosis, a minimum gradient of 3 mmHg in diastole is essential. A gradient of 5 mmHg or more suggests significant stenosis and is generally an indication for surgery.

CORONARY ANATOMY

A basic knowledge of normal coronary anatomy and the blood supply of various

Table 5.9: Coronary arterial division and regional blood supply

<i>Left anterior descending artery (LAD)</i> Septals Diagonals Distal main vessel of LAD <i>Left circumflex artery (LCX)</i> Obtuse marginals (OM) Posterior descending artery (PDA) in 15% <i>Right coronary artery (RCA)</i> Right ventricular branches Acute marginals Posterior descending artery in 85% Posterior left ventricular	Anterior 2/3 of interventricular septum (IVS) Antero-lateral wall of left ventricle Apex Postero-lateral wall of left ventricle Posterior 1/3 of interventricular septum Right ventricular free wall Right ventricular free wall and sometimes posterior interventricular septum Posterior 1/3 of IVS Posterior left ventricular wall with variable area of postero-lateral wall
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Table 5.10: Segments of myocardium and their blood supply

<i>Structure</i>	<i>Usual blood supply</i>	<i>Variant</i>
Interventricular septum, anterior	LAD	RCA (small portions including the apex), circumflex (apex)
Interventricular septum, posterior	RCA (proximal 2/3) LAD (distal 1/3)	Circumflex, LAD (Distal 2/3)
Anterolateral wall	Circumflex, diagonals	Ramus intermedius
Posterior wall	Circumflex, RCA	
Anterolateral papillary muscle	LAD and circumflex	
Posterior medial papillary muscle	RCA	Circumflex
Apex	LAD	RCA, Circumflex
Inferior wall	RCA	Circumflex, Distal LAD

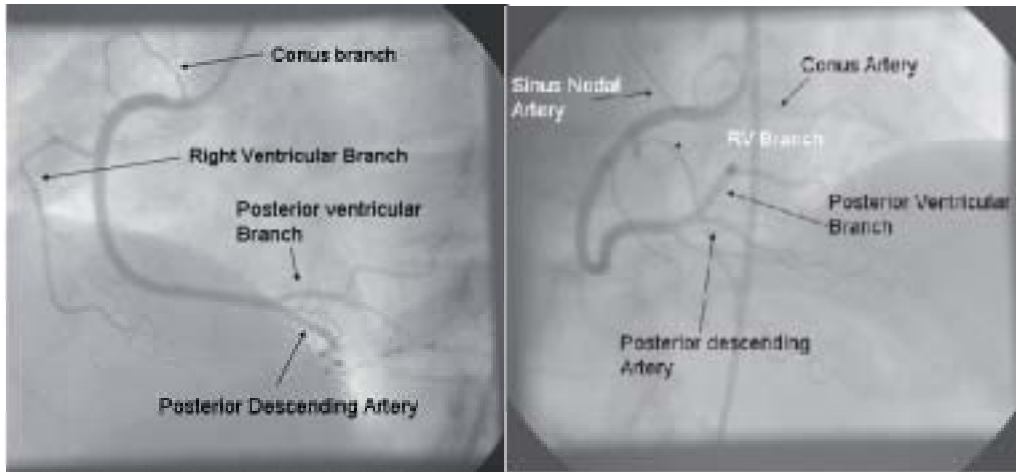


Fig. 5.2: Anatomy of right coronary artery (RCA)

segments of the heart is essential for understanding the various manifestations of ischemic heart disease (Table 5.9).

There are two main coronary arteries – the left (LCA) and right (RCA) coronary arteries. The left coronary artery in turn divides into the left anterior descending (LAD) and the left circumflex (LCX) arteries. The main branches and the areas supplied by each are given in Tables 5.9 and 5.10.

The right coronary artery arises from the right coronary sinus of the aorta and is divided into three segments: proximal (from origin to the right ventricular

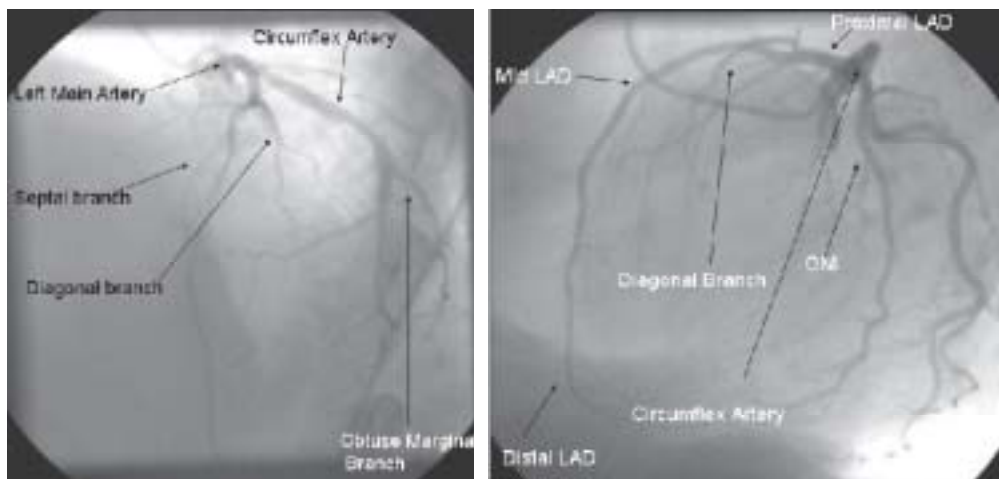


Fig. 5.3: Branches of the left anterior descending (LAD) artery

(RV) branch); mid (from RV branch to the origin of acute marginal (AM) branch); and distal (from AM branch to the termination, including the terminal branches, posterior descending artery (PDA) and posterior left ventricular branch (PLVB)). When the RCA gives rise to the PDA, it is called *dominant*. In 15 per cent of cases the PDA arises from the LCX (Fig. 5.2).

The main trunk of the left coronary artery (LMCA) divides into two main branches – the left anterior descending and left circumflex artery. The LAD is demarcated into three segments – proximal (from origin to the major first diagonal (D1) branch); mid (from D1 to second diagonal (D2)); and distal or apical (the portion distal to D2, which supplies the apical area). Septal branches (S1, S2 etc.) arise perpendicularly throughout its length (Fig. 5.3).

The second major division of left coronary artery courses along the atrioventricular (AV) groove and gives rise to obtuse marginal branches (OMs). The LCX (Fig. 5.4) is divided into two segments – proximal (the portion proximal to the origin of first major OM); and distal (the remaining part of LCX distal to OM1). In 15 per cent of cases, the LCX gives rise to the PDA (*left dominant system*).

The left ventricle is divided into various segments and can be viewed on angiography in angulated views (Fig. 5.6). With the right anterior oblique (RAO) view, the anterior and posterior basal, anterolateral, diaphragmatic, and apical

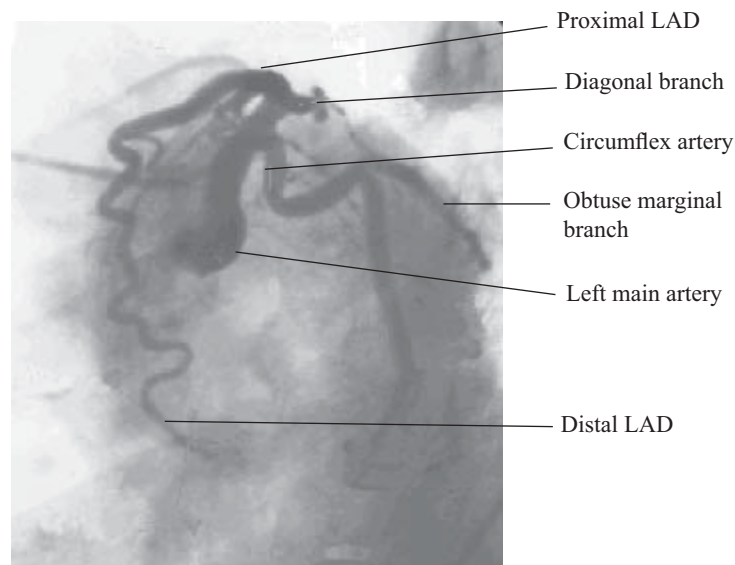


Fig. 5.4: Anatomy of the left circumflex artery (LCX)

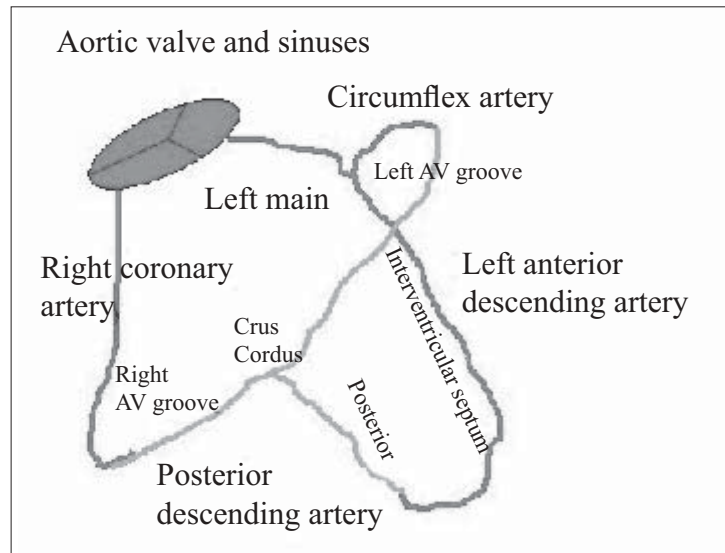


Fig. 5.5: Outline of coronary arterial tree in relation to atrioventricular and interventricular grooves

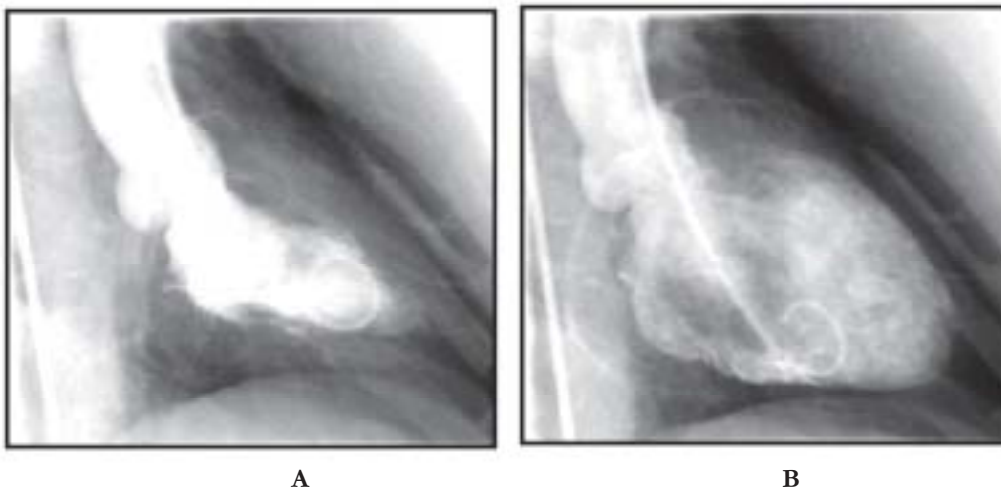


Fig. 5.6: Left ventricular angiogram in systole (A) and diastole (B)

segments can be seen (Fig. 5.7). The anterobasal, anterolateral, and apical segments are supplied by the LAD; and the posterobasal and diaphragmatic segments are supplied by the RCA. In some cases, a variable area of the apex is supplied by the RCA, through its PDA branch.

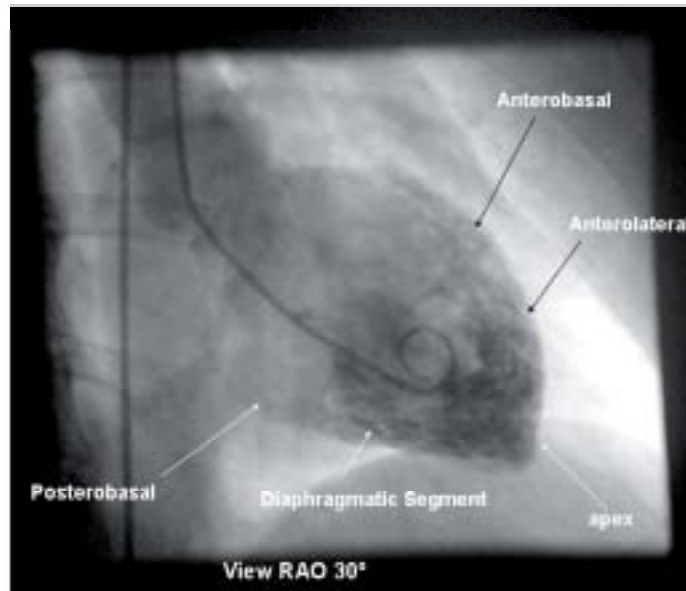


Fig. 5.7: Segmental division of left ventricle (RAO view)

With the left anterior oblique (LAO) view, the entire anterior septum is well profiled. The posterolateral segment and to a variable extent the apex, is also seen (Fig. 5.8). The anterior septum and apex are supplied by the LAD and the posterolateral area is supplied by the LCX.

In conventional angiographic views, some of the segments are not visualized. However, tomographic (cross-sectional) images can be obtained with two-dimensional echocardiography or radionuclide methods. In this tomographic depiction, the basal, mid, distal and apex are depicted from the outer ring to the innermost ring. The septum is divided into anterior two-thirds, supplied by the LAD and posterior third, supplied by the PDA; the lateral wall is divided into anterolateral, supplied by diagonal branches of the LAD, and posterolateral, supplied by obtuse marginal branches of the LCX. The posterior interventricular septum and the posterior wall are supplied by the RCA in 85 per cent, and LCX in 15 per cent of the population.

Coronary arterial lesion assessment

Normally the luminal surface of the coronary artery is smooth and regular. When there is atherosclerosis, there may be a localized narrowing (stenosis), diffuse

narrowing, ectasia or minor irregularities. All of these lesions may have significance in relation to future events. A clinically important lesion is defined as stenosis of at least 50 per cent of the diameter, in a vessel of diameter more than 1.5 mm as measured by calipers.

Areas of narrowing (stenosis) are assessed based on the degree of narrowing and are expressed as 'per cent diameter stenosis' or 'per cent area stenosis'. The common expressions are given below.

<i>Per cent diameter stenosis</i>	<i>Per cent area stenosis</i>
25	44
50	75
70	90
75	95
90	99
100	100

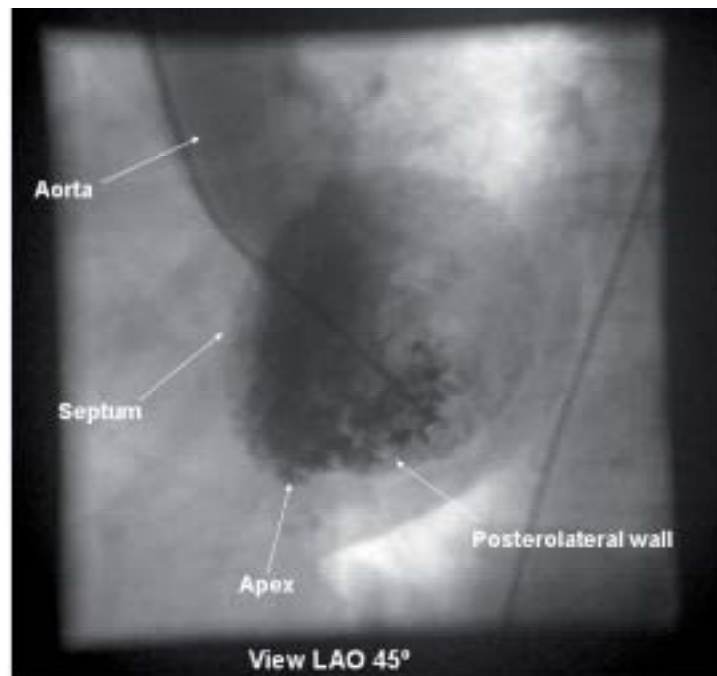


Fig. 5.8: Segmental division of the left ventricle (LAO view)

Fifty per cent diameter narrowing (75 per cent area) is considered significant, and 70 per cent diameter narrowing (90 per cent area) is considered critical. When the vessel is totally occluded, it is considered a 100 per cent block.

The lesions in the coronary artery have been classified into three types based on the morphological characteristics. These are important in planning the type of revascularization and prognosticating the outcome following angioplasty procedures. Table 5.11 gives the classification.

Table 5.11: The American Heart Association/American College of Cardiology Consensus on the characterization of coronary lesion with reference to suitability for traditional balloon PTCA

<i>Type A</i>	<i>Type B *</i>	<i>Type C</i>
Discrete	Tubular	Diffuse (>2 cm)
Concentric	Eccentric	—
Accessible	Moderate tortuosity	Excessive tortuosity
Nonangulated	Moderate angulation	Extreme angulation
Smooth	Irregular	—
Little or no calcium	Mild calcium	—
No occlusion	Occlusion (<3 months in duration)	Occlusion (>3 months in duration)
Not ostial	Ostial	—
No major branch indicated	Bifurcation	Inability to protect a major side branch
No thrombus	Thrombus	Degenerated vein grafts

* Ellis et al suggested modification to stratify type B lesions into type B₁ (one type B characteristic) and type B₂ (more than one type B characteristic) lesions.

6 The Patient's History

It is the province of knowledge to speak and it is the privilege of wisdom to listen.

Oliver Wendell Holmes

The patient's version of the disease is the most important source of information. It directs both the extent of physical examination and the sequence of diagnostic testing. The process of history taking is not mere collection of information. It is at this period that the physician and the patient assess each other. The doctor is obviously eager to reach a diagnosis and to plan the investigation of the patient. The patient, in a subtle and passive manner, will also judge the physician by his appearance and manner. It is at this stage that the 'faith and trust at first encounter' develop. If the doctor does not conduct himself well, the initial interview is summed up as 'distrust at first sight'. The doctor's dress should be clean and dignified. Wearing sports shoes and colourful clothes gives an appearance of casualness and indifference. Also, blank and vacant looks or indulgence in some other activity will lessen the patient's faith. The physician should never appear to be in a great hurry especially during the first encounter.

The interview should take all these patient's concerns and fears into consideration. The history is the source of maximum information related to any illness. It is vitally important to note the patient's main concerns and address them, as the patient is more interested in them than any other smart diagnosis the physician may make. Careful and thoughtful history taking establishes a bond with the patient that may be vital later in securing the patient's acceptance of hospitalization for intensive diagnostic work-up and therapeutic intervention. Some general guidelines are useful.

THE PATIENT'S HISTORY

Table 6.1: The patient's concerns

Anxiety and fear. Do I really need all these tests and procedures? Do I have the disease? Possible death and disability. Will I be able to afford the cost of investigation/treatment? Will they do everything that is necessary and nothing that is unnecessary? Unnecessary investigation and treatment are common today. Outcome of procedures. Complications of procedures. Long queues in the hospitals (for treatment, medication, investigation). Mistakes in diagnosis and treatment are not rare. Should I take a second opinion? Doctors get upset if patients ask for another opinion. Do I need all these medicines? What happens if I stop medication? The doctor didn't spend enough time with me. Has he thought deeply enough about my illness? I am rich and important. Will they overinvestigate and treat? Does he really care about what happens to me?
--

Table 6.2: Patient interview

<i>Patient interview: guidelines to the doctor</i>
The patient is the most important person in the room. Introduce yourself to the patient and the family. There should be no interruptions during the interview or in the physical examination that follows it. Telephone calls, and interruptions by hospital staff entering and leaving the room should be avoided. The patient should have privacy during the interview. Find out who else the patient wants in the room during the examination. The patient should not be interrupted while he talks. Do not give an appearance of jumping to conclusions during the initial evaluation even if things appear obvious. In eliciting family history of sudden death, use tact and caution when a close family member died at the same age as the patient. Avoid being judgmental when patients mention habits of smoking or alcoholism. Do not look disinterested and bored; instead individualize patient evaluations. Never appear to be in a hurry; appear relaxed. Dress in a sober and dignified way.

One should go out of the way to give an appearance of respecting the patient as an individual. But these appearances cannot be feigned for long, unless one genuinely respects people who come for help. The patient and the family should be given separate interviews at least once, to have a complete picture of the illness.

The doctor should introduce himself to the patient and the family. This is particularly applicable to the ward round. Often the patient and the family do not know the name of the doctors even after discharge from the hospital.

CASE RECORD

The case format that is frequently used is described below.

Patient's name:	Hospital No:
Address/telephone number:	
Age:	Sex:
Height:	Weight:
Body Mass Index:	
Presenting complaint:	
Patient's immediate concerns:	
Accompanying symptoms:	
History of risk factors:	
Hypertension	
Diabetes mellitus	
Smoking	
Hyperlipidemia	
Family history of CAD	
Past CAD	
System review:	
Associated disorders of importance:	
Peptic ulcer	
Bronchial asthma/COPD	
Drug history:	
Drug allergies:	
Work details:	
Dietary history:	
Description of a typical daily and weekly routine of the patient:	
Conclusions drawn from history:	
Patient's perception of illness:	
Previous tests done and results:	
Details of previous treatment and response:	
Concerns of the family:	

i) Name and Hospital No: The importance of properly identifying the patient cannot be overemphasized. In busy hospitals and laboratories, mistakes related to patient identity are more common than is realized. Since more than one patient may have the same name, the hospital number should be routinely used.

ii) The importance of height, weight, body mass index: The height of the individual has significance in certain cardiovascular disorders (for example,

THE PATIENT'S HISTORY

Marfan's syndrome). Tall and lean individuals have a proclivity to have mitral valve prolapse.

iii) The importance of age and congenital heart disease: Congenital heart disease is likely until 3 years of age. Rheumatic heart disease is unlikely before the third year. The commonest cyanotic heart disease at birth is D-transposition of great arteries (Table 6.4). After 2 years of age, the commonest cyanotic heart disease is tetralogy of Fallot. Rheumatic heart disease is most common between 10 and 30 years of age. After 40 years, coronary artery disease is more common. Age of onset of heart failure and cyanosis give valuable clues to underlying heart defect in congenital heart disease.

Table 6.3: Definitions of age related terms

Newborn	First week of life
Neonate	First month of life
Infancy	First year
Childhood	1–16 years
Adolescence or teenage	13–18 years
Adulthood	Beyond 18 years
Middle age	50–60 years
Old age	65–75 years
Very old age (octogenarian)	More than 75 years

Table 6.4: Clues to identify underlying cyanotic congenital heart disease from age

<i>Age of onset of cyanosis</i>	<i>Possible diagnosis</i>
At birth	D-TGA Admixture lesions TAPVC Single ventricle DORV Truncus arteriosus Tricuspid atresia Tetralogy of Fallot
After first month	Tetralogy of Fallot
After 2 years	Eisenmenger syndrome due to VSD
After 10 years	Eisenmenger syndrome due to VSD
After 20 years	Eisenmenger syndrome due to ASD

Onset of heart failure gives a useful clue to the diagnosis of underlying cardiovascular disorder. This is particularly valuable in the pediatric age group (Table 6.5).

Table 6.5: Clues to the diagnosis of heart failure from age in congenital heart disease

<i>Age of onset of CHF</i>	<i>Possible diagnosis</i>
On the first day of life	Neonatal heart muscle dysfunction Asphyxia Transient myocardial ischemia Sepsis Hypoglycemia Hypocalcemia Neonatal hematological abnormalities Anemia Hyperviscosity syndrome Neonatal structural abnormalities Congenital tricuspid regurgitation Congenital pulmonic regurgitation Systemic arteriovenous fistula Neonatal heart rate abnormalities Supraventricular tachycardia Congenital complete heart block Neonatal myocarditis
First week of life	Structural abnormalities Critical aortic stenosis Coarctation of the aorta Interrupted aortic arch Hypoplastic left heart syndrome TAPVC Critical pulmonic stenosis PDA (premature infants) Heart muscle dysfunction as above (asphyxia is less likely) Heart rate abnormalities as above Renal abnormalities Renal failure Systemic hypertension Endocrine disorders Neonatal hyperthyroidism Adrenal insufficiency
First 2 months of life	Structural abnormalities Aortic level shunt (PDA, truncus, AP window) Ventricular level shunt (VSD, AV canal, single ventricle) Left sided obstructive lesions as above Atrial level shunt (ASD, non-obstructed TAPVD)

THE PATIENT'S HISTORY

<i>Age of onset of CHF</i>	<i>Possible diagnosis</i>
	Anomalous left coronary arising from pulmonary artery Pulmonary abnormalities/chronic hypoxia (right heart failure) Central nervous system hypoventilation Upper airway obstruction ‘Bronchopulmonary dysplasia’ Heart muscle abnormalities Cardiomyopathy/endocardial fibroelastosis Myocarditis Pompe’s disease Renal and endocrine disorders Hypothyroidism

As a univariate feature, age at onset of symptoms as a powerful factor in the diagnosis of congenital heart disease is illustrated in the following case summary.

Case summary

A 9-year-old girl presented with history of dyspnea. After a physical examination, two-dimensional echo and pulse wave Doppler evaluation, she was diagnosed to have ostium secundum atrial septal defect with left to right shunt and moderate pulmonary arterial hypertension. She was scheduled for surgical closure of ASD two days later.

A day before surgery the patient was presented by a student for a bedside discussion. The student made a clinical diagnosis of ASD but clearly brought out the fact that the child had symptoms of heart failure at the age of 1 month and gradually improved in the next one year. Surgery was postponed and cardiac catheterization and angiography confirmed the diagnosis of atrial septal defect and large ventricular septal defect with hyperkinetic pulmonary arterial hypertension. Obviously the diagnosis of VSD was missed on initial echo evaluation and diligent history taking pointed to this; the mistake was rectified.

Age is a guiding clue not only in the diagnosis as outlined above but also in the management of cardiovascular disease. The age of the child is a very important variable in the management of the ventricular septal defect, which is the commonest of congenital defects.

Age plays a very important role in diagnosing ventricular septal defect (Table 6.6). Spontaneous closure of ventricular septal defect most commonly occurs by 2 years of age but may occur later and is reported in adults. If a new murmur appears after 6 months of age, ventricular septal defect is unlikely.

Table 6.6: Age of child and ventricular septal defect

<i>Age of detection of murmur</i>	
Murmur < 18 hrs of life	Almost never due to isolated VSD
Murmur detected 2–6 weeks	VSD is most likely
2nd or 3rd day (prior to discharge from newborn nursery)	Small VSD with a very rapid decline in PVR; almost never due to isolated small VSD
Later than 4th month	Delayed appearance of murmur means delayed fall in PVR and may suggest large VSD Some other lesion is likely Murmur may have been overlooked in the previous examination or the child was not examined VSD is unlikely
Later than 6 months	
<i>Onset of heart failure</i>	
3rd to 4th month	Most common
Earlier than 1st month	Prematurity, Some other lesion is likely
<i>Timing of surgery</i>	
< 12 months	<i>Indication for surgery</i> Uncontrolled CCF
> 12 months	Qp/Qs > 2:1
<i>Long term result</i>	
Surgery before 2 yrs	Normal PVR on long term follow up
Surgery after 2 yrs	Less predictable fall in PVR

Patients with large ventricular septal defect and pulmonary arterial hypertension due to vascular disease who are inoperable, deteriorate by the second or third decade of life, and most of them may die before the age of 40 years.

iv) Age and valvular heart disease: Mitral stenosis occurring below 17 years of age is called juvenile mitral stenosis. Above 35 years of age, all patients with valvular heart disease require coronary arteriography before they are considered for valve surgery by open heart operation. In any patient with valvular heart disease who is above 35 years of age, if the symptoms are atypical or disproportionately high for the degree of valvular defect, coronary artery disease should be excluded. In patients with valvular aortic stenosis above 40 years of age, almost all male patients have calcified aortic valves. In elderly patients requiring valve replacement, a bioprosthetic valve rather than an ordinary prosthetic valve is the choice.

Prognosis in coronary artery disease is significantly influenced by age. The major studies in coronary artery disease highlighted this. In patients above 80

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years, mortality with open heart surgery is as high as 25 per cent. In the CASS (Coronary Artery Surgery Study) registry data of patients with left main coronary artery stenosis, older patients benefited more with coronary artery bypass grafting (CABG) than with medical therapy. The results are summarized in Table 6.7.

Thus magnitude of the survival gains associated with coronary artery bypass surgery increase with age above 50 years, but again decrease as the age exceeds 75 years. Recent studies showed that thrombolysis in acute myocardial infarction is not only safe but also reduces mortality significantly even in the elderly. Older age is generally considered an incremental risk factor for premature death within 5 years after the coronary artery bypass surgery operation. Patients aged 40–50 years have a 5 year survival of 91 per cent compared to those above 50 years, whose survival is about 81 per cent.

v) Sex and coronary artery disease: Coronary artery disease is rare in premenopausal women. Apart from coronary artery disease, the sex of the patient has important implications in the diagnosis and management of heart disease. Women differ from men in the clinical presentation and the pattern of disease. False positive exercise tests are more common in women. In the CASS registry, 39 per cent of women and 11 per cent of men had normal coronary angiograms. This is despite a typical history of angina and a positive exercise electrocardiogram.

Several studies indicate that the morbidity and mortality of coronary artery disease occur 10 years later in women than in men. But once they develop myocardial infarction, women have higher mortality during the first 30 days after myocardial infarction. This interesting set of observations is frequently quoted. A recent review of English literature from 1966 to 1994 by Vaccarino et al analyzed the reasons for this apparent paradox in women with coronary artery disease.

Table 6.7: Age and coronary bypass surgery

Age	4 year survival with medical therapy	4 year survival with CABG	Percentage gain
< 50 yrs	68%	95%	40%
> 65 yrs	51%	82%	61%
65–69 yrs	67%	81%	22%
70–74 yrs	51%	77%	51%
> 75 yrs	56%	75%	34%

Much of the increased early mortality after myocardial infarction in women is explained by the older age, and the more unfavourable risk characteristics of the women. In the long run, when the differences in age and other risk factors are controlled, women tend to have an equal survival.

vi) Drug history: In the initial interview, it is important to enquire into the nature of treatment or drugs the patient is receiving as it has implications in the diagnosis and management (Table 6.8). A detailed account of drug therapy is of help in proper management of patients with heart disease. These features include the name, dosage, duration of therapy, and compliance with therapy.

Table 6.8: Importance of drug history

Drugs can cause, mask, modify, aggravate, abolish or cure a disease or physical symptoms and signs.
Drugs can induce cardiovascular disease.
Implications in selection of drugs in the light of previous drug therapy.
Drugs may have to be stopped or modified before certain cardiovascular procedures.
Public relation component to the specialist: Check the prescription made by the referring doctor carefully before replacing it.
Drugs taken on the day of examination could have a bearing on the symptoms or test results.

Get specific information about the drug, such as:

Nature of drug
Dosage/spacing
Response to therapy
Improvement
Deterioration
Side effects/toxicity
Duration of therapy
Drug compliance
Drug intake on the day of consultation
Drug monitoring

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Both the pharmacological and trade name should be known. Inappropriate dosage and improper spacing of drugs may be responsible for inadequate response to drugs.

Response to drugs is used as a valuable diagnostic clue in clinical medicine (Table 6.9). The response to the earlier medication helps in the diagnosis and in planning future treatment. For example, improvement with diuretics and digoxin makes heart failure the most likely cause for dyspnea. On the other hand, improvement with bronchodilators suggests bronchial asthma. Angina pectoris responds promptly to sublingual nitroglycerine.

The fever and joint pain of acute rheumatic fever responds promptly to aspirin distinguishing it from rheumatoid arthritis. The chest pain of acute benign pericarditis responds to steroids. Following acute myocardial infarction, chest pain may be due to postinfarction angina or due to pericarditis. Prompt response to steroids suggests pericarditis as the cause of pain. Prompt response to nitrates suggests angina.

Not only improvement, but also deterioration gives valuable clues to the underlying disorder (Table 6.10). The deterioration of patients with pericardial tamponade and constrictive pericarditis with diuretics is well known. Conditions with diastolic dysfunction like hypertrophic cardiomyopathy also deteriorate with the volume depletion of diuretic therapy.

In the management of any specific cardiac defect, one must always elicit the history of drugs which aggravate the underlying disorder. Significant or dramatic improvement occurs when these drugs are discontinued. Sometimes drugs themselves may be responsible for inducing cardiovascular disease (Table 6.11).

Table 6.9: Response to drug(s): diagnostic clues

<i>Drug and response</i>	<i>Most likely diagnosis</i>
Chest pain responds to sublingual nitrate/nitroglycerine	Angina pectoris
Dyspnoea responds to diuretics and digoxin	Heart failure
Acute polyarthritis responds to aspirin	Acute rheumatic fever
Acute chest pain responds to steroids	Acute pericarditis of acute myocardial infarction, idiopathic benign pericarditis

Table 6.10: Drugs aggravating cardiovascular disease

<i>Disease</i>	<i>Drug responsible</i>	<i>Mechanism(s)</i>
CAD Angina or MI	Hydralazine Nifedipine Dipyridamole Aspirin Ergot preparations	Reflex sinus tachycardia increases MVO_2 As above Coronary steal Anemia due to GI bleed Coronary spasm
Valvular disease MS	Vasodilators	Reflex tachycardia with reduced diastolic filling time
AS AR, MR	Vasodilators Vasopressors	Hypotension, syncope ↑SVR, hypertension aggravation of lesion
Systemic hypertension	As above (Bronchodilators like ephedrine, sympatho- mimetic in nasal decon- gestants, cold medications, cough syrups) Steroids NSAIDs Tricyclic antidepressants Excessive use of potent diuretics Antidiabetic agents Betablockers	↑SVR, systemic hypertension Salt and water retention Reflex vasoconstriction Adrenergic excess of hypoglycemia Hypertension in pheochromocytoma due to selective alpha stimulation
Congestive heart failure	Calcium blockers Disopyramide Steroids NSAIDs	Myocardial depression Salt and water retention

Cardiovascular drugs affecting other systems: Drugs could affect systems other than the one that is targeted. A list of cardiovascular drugs that could affect other systems is given in Table 6.12. It is a useful practice to rule out a drug-induced disorder whenever a patient develops new or unusual phenomena (symptoms, signs or a laboratory test abnormality).

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Table 6.11: Drug induced cardiovascular disease

<i>Disease</i>	<i>Causative drugs</i>
1. Systemic hypertension	<p>Sympathomimetic drugs Ephedrine and related drugs Nasal decongestants Cold medications Cough syrups Steroids NSAIDs Estrogens Contraceptive pills Hormone therapy to treat menorrhagia Excessive sodium intake</p>
2. Cardiomyopathy	<p>a) Direct acting Alcohol Bleomycin Adriamycin Phenothiazine Tricyclic antidepressants Emetine hydrochloride Paracetamol Cyclophosphamide Reserpine Catecholamines Corticosteroids Lithium Chloroquine Cocaine Methysergide b) Hypersensitivity Methyl dopa Penicillin Sulfonamides Tetracyclines Phenindione Phenylbutazone Antituberculous drugs</p>
3. Pulmonary arterial hypertension	<p>Aminorex fumarate Monocrotaline or other pyrrolizidine (alkaloids) Oral contraceptives Phenformin</p>

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<i>Disease</i>	<i>Causative drugs</i>
4. Hyperlipidemia	Betablockers Diuretics Alcohol Oral contraceptives
5. Acute myocardial infarction	Ergot preparations Cocaine
6. Prolonged QT	Quinidine Procainamide Amiodarone Disopyramide Sotalol Phenothiazines Tricyclic antidepressants (amitryptaline) Non-sedative antihistamines (terfenadine, astemizole) Drugs enhancing the effects of antihistamines (antimalarials, erythromycin, ketoconazole), liquid protein diets
7. Valvular heart disease	Fenfluramine

Table 6.12: Common side effects of some of the drugs used for heart disease

<i>Disorder</i>	<i>Drugs</i>
Dry cough	ACE inhibitors
Acid peptic disease	Aspirin Reserpine
Hyperkalemia	Potassium sparing diuretics Angiotensin-converting enzyme inhibitors Betablockers
Hypokalemia	Diuretics
Systemic lupus erythematosus	Hydralazine Procainamide
Pulmonary fibrosis	Amiodarone
Bronchial asthma	Betablockers
Hepatic dysfunction	Amiodarone Isoniazid for tuberculous pericarditis
Osteoporosis	Prolonged heparin use
Embryopathy	Warfarin sodium during pregnancy as anticoagulant
Neutropenia	Ticlopidine

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Table 6.13: Drugs masking cardiovascular disease

<i>Condition</i>	<i>Drugs modifying</i>
Systemic hypertension	Antihypertensive therapy
Coarctation of aorta	Antihypertensive therapy
Congestive heart failure	Decongestive therapy
Tricuspid stenosis	Diuretics
Mitral stenosis	Diuretics, betablockers
MVP	Betablockers
HCM	Betablockers
Aortic regurgitation	Vasodilator therapy

Table 6.14: Drug withdrawal crises in cardiology

<i>Withdrawn drug</i>	<i>Consequence</i>
Antihypertensive drugs (particularly clonidine)	Hypertensive crisis
Betablockers in CAD	Unstable angina or acute MI
Calcium blockers in CAD	Unstable angina or acute MI
Diuretics	Heart failure or pulmonary edema
Betablockers in tetralogy of Fallot	Hypoxic spells
Oral anticoagulants for prosthetic valves	Thrombotic occlusion of the prosthetic valve
Aspirin following PTCA	Risk of abrupt closure
Heparin following PTCA	Risk of abrupt closure
Heparin or oral anticoagulants following intracoronary stenting	Acute stent thrombosis
Aspirin in unstable angina with severe coronary disease prior to bypass surgery	Acute myocardial infarction

Drugs masking cardiovascular disease: Hypertension or coarctation of the aorta can be masked by antihypertensive drug therapy (Table 6.13). The physical signs in tricuspid stenosis like prominent *a* wave and elevated JVP may be modified by diuretic therapy. The mid-diastolic murmur of mild mitral stenosis may be obliterated by the bradycardia induced by betablockers. Betablockers may also mask any signs of mitral valve prolapse.

Drugs and coronary artery disease: In the functional evaluation of patients with angina, it is important to check whether the angina is in the presence or absence of drug therapy. Patients who are meeting a new doctor have a habit of stopping

Table 6.15: Drugs and other factors that alter the anticoagulant response to warfarin

<i>Prolongs prothrombin time</i>	<i>Reduces prothrombin time</i>
<u>Pharmacokinetic</u> <i>Increases warfarin levels</i> Phenylbutazone* Sulfinpyrazone* Metronidazole* Cotrimoxazole* Erythromycin* Fluconazole* Miconazole* Nafcillin Cimetidine* Omeprazole* Amiodarone* Disulfiram† <u>Pharmacodynamic‡</u> Clofibrate* Low vitamin K intake* Malabsorption Liver disease Hypermetabolic states Coagulopathies <u>Mechanism not established</u> Isoniazid* Propafenone* Propranolol* Piroxicam* Acetaminophen† Anabolic steroids† Aspirin† Chloral hydrate† Ciprofloxacin† Itraconazole† Quinidine† Phenytoin† Simvastatin† Tamoxifen† Tetracyclines† Influenza vaccine†	 <i>Decreases warfarin levels</i> Cholestyramine* Barbiturates* Rifampicin* Carbamazepine* Griseofulvin* Dextropropoxyphene† High vitamin K intake* (certain vegetables, nutritional supplements) Chlordiazepoxide* Sucralfate* Dicloxacillin†

* Highly probable interaction

† Probable interaction

‡ No effect on warfarin levels.

the drugs they were on like betablockers, diuretics or antihypertensives. This may precipitate a critical state.

Patients undergoing coronary bypass surgery (CABG) are often already taking betablockers or calcium channel blockers which have to be continued before surgery. Sudden withdrawal may precipitate unstable angina or myocardial infarction (Table 6.14). Digoxin is usually stopped 36 hours before operation.

Drugs and valvular heart disease: Diuretics mask congestive heart failure and the fourth or third heart sound may be diminished or absent. In tricuspid stenosis, the jugular venous signs and the tricuspid diastolic murmur may decrease or disappear. Even at cardiac catheterization, tricuspid stenosis may be underestimated or may be missed as a result of excessive diuretic therapy. It is for this reason, that all patients with rheumatic heart disease should have their diuretics stopped prior to cardiac catheterization, unless essential. In patients fully digitalized, the diagnosis of atrial fibrillation may be missed as the heart may be normal and relatively regular. In patients on digitalis, the diagnosis of atrial fibrillation may be missed unless one exercises the patient to bring out the rapid and irregular rates. Almost all the patients after prosthetic valve insertion are on oral anticoagulant therapy for life.

Drugs prescribed by the referring doctor: When the patient is prescribed certain drugs, one must make a careful note of all the drugs with the trade names. The specialist should not change the drugs (this includes the trade names) unless essential. Inadequate attention to this is likely to induce misunderstandings between the specialist and the referring doctor. The patient is likely to feel that the original drugs were wrongly prescribed by the referring doctor.

Drugs on the day of consultation: Many patients have the habit of stopping the drugs before visiting a new doctor. Most argue that when they go to the new doctor, the drugs will be changed anyway. When patients are asked to come for the next visit after one month, they interpret it as having to take the drugs only for one month. Some patients believe that they are under unnecessary medication and stop the drugs to see the effect on blood pressure. If the patient's blood pressure is normal on the day of consultation the patient concludes that he may not need drugs for his hypertension and the high blood pressure is 'cured'. Typically, a patient with systemic hypertension may not take the drugs on the day he visits the doctor. The blood pressure will naturally be high and the doctor obligingly

steps up the dosage of antihypertensive agents. This may result in postural hypotension or even frank syncope. The physician finds it puzzling as the blood pressures are high. He may consider another cause for syncope and extensive and costly diagnostic work-up is unnecessarily resorted to.

INCREMENTAL RISK FACTORS

Information from the history, independent of stress testing may be of value in predicting ischemic events. Clinical variables predicting future ischemic events are:

- Angina prior to infarction
- History of symptomatic hypertension
- Diabetes mellitus
- Peripheral vascular disease
- Anterior Q wave first MI
- Heart failure on admission

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Patients with 5 or 6 of these variables had a one year reinfarction rate of 23 per cent compared with 4 per cent in those with none or one of these factors, 5.5 per cent with two of these factors, 8 per cent with three of these factors, and 15 per cent with four of these factors. The ability to predict recurrent myocardial infarction is of importance since it has been shown that recurrent myocardial infarction is one of the most important predictors of subsequent death after infarction.

Incremental risk factors in cardiac surgery

The multiple variables that interact with one another to influence the results of cardiac surgery are called incremental risk factors. Awareness of these factors is useful in decision making in individual patients. Risk assessment of a patient with coronary artery disease, likely to undergo coronary artery bypass graft surgery is given below as an example.

The incremental risk factors for hospital death in CABG are:

- Age > 60 years
- Frank diabetes mellitus
- Systemic hypertension
- Severity of angina

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- Grade of dyspnea
- Female sex
- Unstable angina
- Emergency operation
- Skill of the surgeon

The value of considering this information is that even at the stage of history taking, the risk of interventions like cardiac surgery can be predicted. All the information listed is obtainable from history.

It is useful to follow a checklist in history taking. Table 6.16 gives the list of symptoms to be checked. Though using a checklist gives an appearance of artificiality with regard to history taking, it is better to be thorough. A methodical approach to history taking without a checklist comes only after years of practice. Even then, the senior physician is actually using a mental checklist. The junior student should not hesitate to use the checklists till he has gained enough experience to use a mental one.

Table 6.16: Cardiovascular history checklist

<i>Coronary artery disease</i>	<i>Valvular heart disease</i>	<i>Congenital heart disease</i>	<i>Cardiomyopathy</i>	<i>Prosthetic valves</i>
Risk factors	Fever	Consanguinity	Family history	Age
Family history	Sore throat	Cyanosis	Drug history	Sex
Hypertension	Arthralgia/	Spells	Alcohol	Pregnancy plan
Diabetes	arthritis	Dyspnea	Respiratory type illness	Bleeding tendency
Smoking	Dyspnea	Syncope	Dyspnea	Food intake
Hyperlipidemia	Palpitation	Edema	Syncope	Vitamins (K)
Female hormones	Syncope		Chest pain	Diarrhea
Chest pain	Edema		Systemic disorder	Antibiotics
Dyspnea	Chest pain			Aspirin/analgesics
Palpitation	Prophylactic			Prothrombin time
Syncope	Penicillin			Peripheral embolism
Weakness				Fever
				Dyspnea
				Syncope
				Edema
				Valve noise

The best thing one can say about history is that there can never be enough of it.

"Opportunity is often lost because we are broadcasting when we should be tuning in."

Anonymous

CONTROVERSIES AND PERSONAL PERSPECTIVE

It is clear that increasingly, taking a thorough history has become less of a priority for most doctors. First, there is less time available for physician-patient direct contact as a result of the demands placed on a physician's time. Frequently, a physician extender or nurse obtains part or all of the history to reduce the time burden on the doctor. Under the putative heading of 'efficiency', the time and importance given to detailed, thorough interview has been markedly reduced. Second, with advances in technology, history taking and physical examination have been partially supplanted by rapid use of bedside tools such as the 2-D echocardiogram, treadmill or dobutamine thallium test, Holter recording or even diagnostic angiography or electrophysiologic study.

This development is unfortunate. The history is the most important way to create a patient-physician bond and is precious and irreplaceable. The task should not be delegated to a physician extender, ideally, except for such perfunctory aspects as the medication and dose, risk factors, and minor details. It is absolutely essential to directly query the patient in depth for the principal symptoms such as chest discomfort, loss of consciousness, or dyspnea. Only in this way will there be the optimal accuracy and relationship building that are critical to treatment. Also, imprudent use of advanced diagnostic technology is not only expensive but can uncover findings that, while accurate, have no bearing on the patient's symptoms. The detailed history will preempt or properly guide the use of more refined diagnostic tests. The history is the most cost-effective tool that one can use to soundly lay the foundation of patient evaluation.

Although history taking has lost the pride of place in a diagnostic work-up in the past decade, it will be increasingly relied on in the future due to its relatively low cost and pivotal importance in guiding more diagnostic and therapeutic decision making.

7 Evaluation of Chest Pain: Diagnostic and Therapeutic Implications

There is a disorder of the breast marked with strong and peculiar symptoms, considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned more at length. The seat of it, and sense of strangling, and anxiety with which it is attended, may make it not improperly called angina pectoris... Those who are afflicted with it are seized while they are walking and more particularly when they walk soon after eating with painful and most disagreeable sensations in the breast which seem as if it would take their life away, if it were to increase or to continue. The moment they stand still all this uneasiness vanishes.

William Heberden

This masterly description of effort angina by William Heberden over two hundred years ago has not been improved upon. Chest pain is the most common clinical expression of coronary artery disease. The interpretation of chest pain to this day remains the most important method of recognizing coronary artery disease. There are however many reasons for failure to recognize coronary artery disease from its symptomatic expressions. Myocardial infarction is unrecognized in 12 per cent and unaccompanied by chest pain in 25 per cent of patients. The more recent and realistic description of the problem is by Harrison and Reeves.

Discomfort in the chest whether actually painful or not, is usually due to a relatively innocent or a potentially grave disorder. Unfortunately there is no parallelism between the severity of the disease and the gravity of its cause.

Case summary

A 39-year-old airline engineer presented with substernal chest discomfort related to food. Each episode lasted 5–10 minutes and was relieved by belching. He was a chronic smoker

and had a history of acid peptic symptoms in the past. He was admitted in CCU for observation. Physical examination was unremarkable and the serial ECGs were normal. The cardiac enzyme levels were normal. The pain was considered atypical for myocardial ischemia by the treating cardiologist but he was prescribed diltiazem, nitrates and H₂ antagonists. During the two days of hospital stay he had two brief episodes of burning chest discomfort partially relieved by nitrates. He was discharged after 2 days of hospital stay and was advised to come back for an exercise test after 1 week. Three days after discharge, he developed substernal pain and collapsed. He was 'brought dead' to the emergency room. He is survived by a 32-year-old wife and a 7-year-old son suffering from severe rheumatic mitral regurgitation.

The factors related to the patient, the physician, medical education, medical literature and other issues conspire together in this distressingly common failure to recognize myocardial ischemia or infarction. Pain is a warning signal. The reaction to a warning is a matter of attitude of the person, affordability, and the culture of the society we live in. The patient should first have pain and feel the need for immediate medical help. When the pain is at unusual sites or is mild and vague, the patient tends to neglect it altogether and may never reach a physician. Even when they reach a medical care facility, there is no guarantee that all that needs to be done will be done. The professional inadequacy of the doctors is easily understood if one looks at their training. Neither medical students nor postgraduates are trained properly in the clinical evaluation of patients with coronary artery disease (CAD), as patients with coronary artery disease are not 'examination cases'.

APPROACH TO A PATIENT WITH CHEST PAIN

A patient with acute chest pain needs a systematic approach that combines the diagnostic considerations and therapeutic options. The table below gives the suggested approach.

If cardiac, is it due to

- Myocardial ischemia?
- Pericarditis?
- Aortic dissection?
- Acute pulmonary embolism?
- Mitral valve prolapse?
- Hypertrophy disorders of myocardium?

If due to myocardial ischemia, is it due to

- Hypertrophy disorders of myocardium?
- Coronary artery disease?

If due to CAD is it

- Acute myocardial infarction?
- Unstable angina?
- Chronic stable angina?
- Vasospastic angina?
- 'Mixed angina'?

If non-cardiac, is it

- Esophageal?
- Gastroesophageal reflux?
- Nutcracker esophagus/ esophageal spasm
- Following vomiting?
- Musculoskeletal?
- Costochondritis (Tietz's syndrome)
- Increased muscle tension?
- Osteoarthritis of cervical/ dorsal spine?
- Tumors of breast?
- Hyperventilation/psychiatric?
- Panic disorder?
- Depression?
- Somatization disorder?
- Acute peptic ulcer syndrome?
- Acute pancreatitis?
- Acute cholecystitis?

Causes of chest pain

Chest pain may be due to extrathoracic and thoracic structures like tissues of the neck, or thoracic wall, thoracic muscles, cervicodorsal spine, costochondral junctions, breasts, sensory nerves, spinal cord; intrathoracic structures like aorta, pulmonary artery, bronchopulmonary tree, pleura, mediastinum, esophagus and diaphragm; subdiaphragmatic organs like stomach, duodenum, pancreas, and gall bladder and liver.

Structures responsible for chest pain are:

A. *Intrathoracic*

Heart
Aorta
Pulmonary artery
Pleura
Mediastinum
Bronchopulmonary tree
Esophagus
Diaphragm

B. *Extrathoracic*

Skin and subcutaneous tissue of thorax
Soft tissues of the neck
Cervical spine/dorsal spine
Costochondral junctions
Breasts
Sensory nerves
Spinal cord

C. *Subdiaphragmatic*

Duodenum
Stomach
Gall bladder
Liver
Pancreas
Spleen
Non-organic

If the physician has a complete knowledge of the varied clinical manifestations of each these disorders, he will easily be able to differentiate each type of pain from the other. Certain features of the pain help distinguish cardiac from musculoskeletal causes of pain as listed in Table 7.1.

MYOCARDIAL ISCHEMIA

EVALUATION OF CHEST PAIN DUE TO MYOCARDIAL ISCHEMIA

Doubt is not a pleasant condition, but certainty is absurd.

Voltaire

Appropriate management of a patient presenting with chest pain needs a systematic approach as outlined (Table 7.1). The central theme is to be overcautious in the approach to the problem. As no single feature in chest pain is diagnostic, integration of all the available data helps in the diagnosis. Given a subset of clinical presentation, the probability changes significantly by the associated risk factors for coronary artery disease. A risk factor may be defined as any habit or trait that can be used to predict the probability of developing that disease in an individual. It is a causative agent or condition that can be used to predict an individual's probability of developing disease.

The risk factors for coronary artery disease are:

- Family history of premature coronary artery disease
- Tobacco smoking
- Systemic hypertension
- Elevated plasma cholesterol
- Diabetes mellitus
- Ageing

Table 7.1: Features of cardiac pain

<i>Feature</i>	<i>Cardiac, if</i>	<i>Non-cardiac, if</i>
Location	Diffuse, substernal with radiation	Localized, left inframammary
Quality	Dull, deep, aching, pressing	Shooting, sharp, cutting
Intensity	Mild to moderate with gradual fluctuation	Rapidly fluctuating
Duration	Minutes or hours	Seconds or fluctuating
Precipitated by	Effort, emotion, cold	Posture, respiration
Relieved by	Brief rest and nitroglycerine	Lengthy rest and most other measures

- Male sex
- Obesity

The prior probability of coronary artery disease is high in patients with one or more of these risk factors. The characteristic features of chest pain must be interpreted in the light of the prior probability that a person of a given age and sex, with a particular past medical history would have such a cause of chest discomfort. For example, if a 50-year-old man with coronary risk factors like smoking, hypertension and diabetes mellitus presents with even atypical chest discomfort, coronary artery disease should be considered. On the other hand, if a 25-year-old woman with no risk factors or use of oral hormones comes with typical chest discomfort seemingly suggestive of angina, coronary artery disease is unlikely.

Before we explain the value and limitation of each of the features that help evaluate chest pain, we need to define certain terms.

The history and mechanism of various clinical subsets of angina

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Effort stable angina: Angina usually occurring at a predictable level of exercise, relieved gradually with cessation of exercise (mechanism: disproportionate increase in myocardial oxygen requirements, demand ischemia).

Unstable angina: Rest angina; new onset of accelerated angina; rapidly progressive worsening stable angina with or without changes in character, severity, or duration (mechanism: recurrent nonocclusive coronary artery thrombi; supply ischemia).

Postinfarction angina: Recurrence of angina at rest or during activity within 30 days of infarction (mechanism: similar to primary unstable angina).

Vasospastic or Prinzmetal angina: Rest angina only, or rest angina and angina only at a very high level of exercise; history of 'cyclical angina'; often due to focal spasm of the epicardial coronary arteries. There may be history of Raynaud's phenomenon or migraine.

Mixed angina: Variable exercise threshold (mechanism: combined demand and supply ischemia).

Postprandial angina: Angina precipitated by meals (mechanism: supply ischemia).

Nocturnal angina

- Occurring within 1–2 hours after assuming recumbent position (mechanism:

demand ischemia).

- Occurring after going to sleep, usually in the early morning hours (mechanism: supply ischemia).

Walk-through angina: Angina occurring at the beginning of exercise and resolving despite continuing same level of exercise (mechanism: supply ischemia).

Linked angina: Angina occurring with esophageal reflux, gall bladder, or other visceral disease.

Syndrome X or microvascular angina: History is similar to that of effort stable angina. (Diagnosis can be made only after coronary angiography which demonstrates normal epicardial coronary arteries; mechanism not certain; impaired coronary vasodilatory reserve is a possibility).

Atypical chest pain syndrome: Chest pain of variable character, duration and location; localized or diffuse; not precipitated or influenced by physical activity (not due to myocardial ischemia; the mechanism of pain is unknown; decreased pain threshold and anxiety may be associated).

Pathophysiology of discomfort due to myocardial ischemia

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Ischemic myocardium releases adenosine, bradykinin, histamine and serotonin. Acidosis and elevated potassium may trigger release of these substances. The sensory end plates of the cardiac sympathetic nervous system are stimulated by these substances. The endplates are the receptors of a network of unmyelinated fibres that lie between cardiac muscle fibres. They are also found around coronary vessels. They form the cardiac plexus and ascend to the sympathetic ganglia. The endplates transmit the pain impulses to the corresponding spinal ganglia, through the spinal cord to the thalamus and the cerebral cortex. The discomfort is referred to the peripheral dermatomes that supply afferent nerves to the same segment of the spinal cord as the heart. A common pool of secondary neurons can be stimulated by somatic and visceral afferent impulses. When the visceral stimuli are excessive, the surrounding intermediate neurons that are receptors for somatic impulses may be excited, resulting in discomfort in the cutaneous location. This is called the phenomenon of *irradiation of impulses*. The connections to the brachial plexus and cervical nerve roots explain the pain in the medial aspect of the arm and neck, respectively.

There is a wide variation in pain perception in myocardial ischemia. The autonomic denervation in diabetes may explain the frequent occurrence of silent ischemia (ischemia without pain) in diabetic patients. Patients with silent ischemia may have altered central modulation of pain perception. Patients with silent ischemia have a higher dental pain threshold, and the intensity of pain is less even when the threshold is reached. Studies have shown that the angina is less likely if the exercise induced endorphin levels are high.

A systematic evaluation of chest pain should take into account the following factors (Table 7.1):

Onset: A dramatic or crushing type of pain is a feature of acute aortic dissection. The pain is most severe at the time of onset but gradually decreases in severity over a period of time. On the other hand, the pain of acute myocardial infarction is sudden in onset, continues in severity for 1–6 hours and subsides, or may wax and wane. The waxing and waning pain correlates with subtotal occlusion of infarct related artery by a thrombus with superimposed spasm, spontaneous recanalization, reocclusion, or recruitment of collateral. The discomfort of angina is usually gradual in onset. Acute coronary syndromes (a spectrum encompassing unstable angina, non-QMI and acute MI) tend to occur most commonly in the early morning hours or at the time of waking up in the morning. The pathophysiological mechanisms underlying this circadian periodicity for acute coronary syndromes are listed below:

- Increased plasma catecholamines
- Increased cortisol

Table 7.2: Factors in evaluation of chest pain

Onset/time of occurrence
Duration/time to treatment
Site
Radiation
Character
Severity
Relation to physiological act
Relief with rest and drugs
Accompanying symptoms:
Symptoms related to loss of contracting
myocardium and conducting tissue
Symptoms of autonomic excess
Sympathetic excess
Parasympathetic excess

- Increased blood pressure and heart rate
- Increased platelet aggregation
- Increased tissue plasminogen activity
- Increased fibrinolytic inhibitor (PAI-I)

Site and radiation: The commonest site of ischemic pain is substernal and a similar area over the back with radiation to the left arm. The left arm radiation is because the same nerves supply the arm and the heart. The site of pain is variable and can occur anywhere, from the upper abdomen to the lower jaw and can occur on either side of chest and may radiate to the right or left arm, to the neck or jaw. In patients with a pre-existing chronic disorder, the site of referral is usually to the site of previous pain. The discomfort seldom extends above the jaw or below the umbilicus.

The primary pain may be altogether absent with the site of referral as the only site of pain. For example, patients with peptic ulcer have their pain of acute myocardial infarction referred to the epigastrium.

Case summary

A 56-year-old practising cardiologist started having pain in the neck and back variably related to exertion. He had symptoms attributable to cervical spondylosis for the past 10 years. He interpreted the recent symptoms to the same disorder and ignored the symptom. When the pain became more frequent, he started using analgesics and cervical collar. He had frequent pain on exertion and also at rest with occasional nocturnal pain for 2 months. He drove about 300 km himself to attend his daughter's marriage and drove back. His symptoms progressively worsened and he sought an orthopedic consultation. The orthopedic surgeon noticed edema of both lower limbs and suggested an ECG and consultation with a cardiologist. The ECG revealed extensive anterior myocardial infarction of uncertain age and ischemia in other territories. He was hospitalized and was found to have congestive heart failure and recurrent angina. In view of recurrent angina and episodic pulmonary edema, an intra-aortic balloon pump was inserted. His condition improved in the next few days but angina recurred the moment intra-aortic balloon was discontinued. Coronary angiogram revealed severe triple vessel disease and severe left ventricular dysfunction with an ejection fraction of 25 per cent. An emergency surgery was planned, but he had an arrest before he was shifted to the operating table. Attempts at emergency revascularization were unsuccessful. He expired.

This case summary illustrates the treacherous deception of the presenting symptoms of coronary artery disease. Even a cardiologist who deals with this disorder all the time is deceived into believing for several weeks that it was cervical spondylosis.

Table 7.3: Typical sites of pain due to myocardial ischemia

<i>Common</i>	<i>Uncommon</i>
Substernal	Elbow
Back (interscapular)	Hand
Jaw	Right side of chest
Throat	Right arm
Left arm	Head
All over anterior chest	
Both shoulders	
Both arms	
Upper abdomen	

As the site of pain is variable and can be atypical in location, mistakes in diagnosis are common (Table 7.4).

The terms typical and atypical angina are often used in clinical practice. Substernal location, precipitation by exertion and relief by nitroglycerine were required for the diagnosis of definite angina in the coronary artery surgery study (CASS), and this is considered typical angina. Table 7.5 summarizes the features of typical and atypical angina.

Table 7.4: Typical sites for pain due to myocardial ischemia

<i>Site of pain</i>	<i>Mistaken for</i>
Upper abdomen	Acid peptic disease
Neck pain	Cervical spondylosis
Back pain	Muskuloskeletal pain
Jaw pain	Dental pain
Throat pain	Throat problem
Right sided chest pain	Non-cardiac problem
Left arm pain	Spondylosis
Right arm pain	Spondylosis
Bilateral shoulder/arm pain	Spondylosis
Substernal pain	Esophagitis/acid peptic disease
Anterior chest pain	Esophagitis/acid peptic disease

Table 7.5: Characteristics of typical and atypical angina

<i>Typical angina</i>	<i>Atypical angina</i>
Substernal	Location in the left chest, abdomen, back, or arm in the absence of mid chest pain
Heavy, squeezing, or burning	Sharp, fleeting
Precipitated by exertion or emotion	Repeated or very prolonged Unrelated to exercise
Prompt relief by rest or nitroglycerine	Not relieved by rest or nitroglycerine Relieved by antacids Shortness of breath or palpitations without chest pain

Duration of pain: If the duration of pain is less than a minute, the pain is unlikely to be of cardiac origin. In classic stable angina, the pain usually lasts 3–5 minutes, generally does not exceed 15 minutes and almost never lasts beyond 30 minutes. In chronic stable angina, the duration of pain in a given patient is constant. Any change in the duration of pain should be considered suggestive of progression of the disease to either unstable angina or acute myocardial infarction. Angina induced by emotion usually lasts longer and may be misinterpreted as change in chronic stable angina. Prolonged pain by definition means pain lasting longer than 30 minutes and usually suggests unstable angina or acute myocardial infarction. Persistent or continuous pain for many days or weeks is obviously not consistent with pain of myocardial ischemia or infarction.

Two aspects of the duration are important: the time from the onset of pain to hospitalization, and the duration for which the pain lasts. In an evolving myocardial infarction, if the pain is less than 6 hours in duration, interventions to recanalize the occluded coronary artery are applicable. The recanalization rates with streptokinase in the first hour are 75 per cent and are only 40 per cent in the sixth hour. Significant reduction in mortality occurs if the patient reaches the hospital early and thrombolytic therapy is given (Table 7.6).

The crucial importance of the time factor in the setting of myocardial infarction should be stressed at all relevant professional and public fora.

In a very useful study, Cox and associates have shown that patients with acute myocardial infarction who are otherwise eligible for thrombolytic therapy, have a high likelihood of developing an infarct even if they are free from chest pain

Table 7.6: Importance of early thrombolytic therapy in acute myocardial infarction

<i>Time from onset</i>	<i>Reduction in mortality</i>
< 1 hr	47%
< 3 hrs	23%
3–6 hrs	17%
6–9 hrs	11%

before lytic therapy is begun. Lytic therapy appears to be safe in this group of patients as in those with continuing chest pain; also subsequent left ventricular function is better. Thus, it is reasonable to consider thrombolytic therapy for patients with ST elevation and ischemic chest pain of less than 4 hours duration who become free of chest pain with nitrates, morphine or betablockers.

Character of pain: The pain is usually described as heaviness, constricting or choking. However the character is highly variable and should not be relied upon. Burning pain is common and could be confused with acid peptic disease. A diffuse burning pain all over the anterior chest, is highly suggestive of acute myocardial ischemia. A significant number of patients come with a 'difficult to describe' type of discomfort. This non-specific and vague feeling is particularly associated with myocardial ischemia. The terms commonly used by people vary from country to country and in the same country from place to place. The student and practitioner should be aware of these descriptions in the community in which they practise medicine.

The terms commonly used to describe the chest discomfort of myocardial ischemia are:

- Heaviness
- Squeezing
- Gripping
- Compression
- Burning
- Gas
- Acidity
- Uneasiness
- Uncomfortable feeling

Severity of pain: The severity of pain is highly variable and is an unreliable feature in the evaluation of chest pain. It is understandable when we know that the pain of myocardial ischemia or infarction varies from absence of pain to extremely severe pain. In many patients, the pain is so mild that they fail to seek medical opinion. The classic descriptions of pain of myocardial infarction as one of the most severe pains next only to the pain of acute pancreatitis, perforated peptic ulcer and aortic dissection is true only in a minority of patients. Narins et al in a prospective study evaluated the relationship between pain tolerance and several clinical variables and ischemic test variable including stress thallium scintigraphy. Patients with high pain threshold had less angina prior to the index coronary event but more inducible ischemia after the event. There was no difference in cardiac event rates (death or non-fatal myocardial infarction) between the low and high pain threshold groups. Patients with high pain threshold are usually younger.

Aggravating and relieving factors

Relation to a physiological act: In the evaluation of pain anywhere in the body, this feature is the most valuable. For example, the pain of peptic ulcer is related to food and movement of the body part aggravating the musculoskeletal pain. The pain of classic angina is related to exertion and is relieved by rest. Unlike musculoskeletal pain, where the pain starts flush with the beginning of exercise, the pain of angina starts some time after the exercise and subsides some time after the cessation of exercise. In other words there is always a lag period between the beginning of exercise to the onset of pain, and cessation of exercise to relief of angina. Pain that starts a few minutes or hours after the cessation of exercise is also not due to angina. Apart from exercise, other factors can precipitate angina like exposure to cold or hot weather, food, bending forward, dreams, and emotions.

Myocardial oxygen consumption depends upon the following determinants:

- Heart rate
- Blood pressure
- Myocardial contractility
- Wall tension

All the above factors precipitate myocardial ischemia either by increase in demand or by decrease in supply of coronary flow. Chauhan et al in a study demonstrated angina pectoris precipitated by acid stimulation of esophagus in

Table 7.7: Precipitating factors for angina

<i>Factor</i>	<i>Mechanism</i>
Exercise	Heart rate, blood pressure
Emotion	Excessive catecholamines Heart rate Blood pressure
Cold environment	Vasoconstriction Blood pressure Coronary vasoconstriction with diminished coronary reserve
Hot environment	Vasodilatation Reflex tachycardia ↑Cardiac output
Food	Splanchnic vasodilatation ↑Cardiac output
Reflux esophagitis	Linked angina due to acid stimulation
Dreams	Emotion Precipitation of arrhythmia
Supine position	Increased venous return Increased end-diastolic volume of ventricles Increased wall tension
Bending forward	? Reflex coronary vasoconstriction ? Compression of epicardial coronaries
Fever especially with chills and rigors	Vasodilatation and reflex tachycardia Chills and rigors are similar to muscular exercise
Straining at stool or micturition	Like Valsalva straining phase Fall in central aortic pressure Reflex tachycardia

patients with angiographically proven coronary artery disease. Acid stimulation of esophagus failed to elicit angina in a group of patients with heart transplantation, proving the neural reflex mechanism. As this angina is related closely to esophageal stimulation by acid, this is termed as *linked angina*. Marchant et al studied the effects of cold weather in patients with angina pectoris. They showed that exposure of patients to cold weather causes peripheral vasoconstriction and an increase in blood pressure, particularly at submaximal exercise level. In spite of rise in blood pressure, increase in myocardial oxygen consumption may not occur if the heart rate is reduced by the baroreceptor mechanism.

Table 7.8: Clinical situations where drugs can precipitate angina

<i>Clinical state(s)</i>	<i>Mechanism</i>
<i>Bronchial asthma</i> Sympathomimetics Ephedrine Alpha-2 stimulants (Salbutamol)	Tachycardia Hypertension ↑Cardiac output ↑Myocardial contractility
Steroids Salt and water retention Hypertension	↑Cardiac output
<i>Nasal allergy</i> Decongestants/sympathomimetics	Hypertension, tachycardia
<i>Systemic hypertension</i> Hydralazine Nifedipine	Sinus tachycardia Sinus tachycardia Coronary steal phenomenon Hypotension
<i>Diabetes mellitus</i> Insulin and oral drugs	Hypoglycemia Tachycardia Hypertension with adrenergic excess
<i>Coronary artery disease</i> Nifedipine	Reflex tachycardia Coronary steal
Nitrates/nitroglycerine	Mitral valve prolapse or hypertrophic cardiomyopathy mistaken for CAD
Aspirin	Anemia due to GI bleed
<i>Hypothyroidism</i> Thyroxine	Excessive dosage Tachycardia ↑Cardiac output
<i>Joint disease</i> NSAIDs Steroids	Fluid and salt retention Hypertension Anemia due to GI bleeding
<i>Peptic ulcer</i> Parasympatholytics Antacids	Tachycardia Interfere with absorption of anti-anginal agents
<i>Depression/chronic pain</i> Tricyclic anti-depressants	Ventricular arrhythmias Hypertension

In patients with abnormal baroreceptor function, the reduction in heart rate may not occur in response to cold induced increase in blood pressure. The rate pressure product increases resulting in ischemia at lower work load. This mechanism explains the variability in clinical behavior between 'cold tolerant' and 'cold intolerant' patients with angina. The baroreceptor function is abnormal in cold intolerant patients.

The accompanying chills and rigors in fever act like muscular exercise and may precipitate a myocardial infarction in a patient with significant angina or unstable angina.

Certain drugs can precipitate or aggravate the existing angina by increasing the heart rate, blood pressure or cardiac output (Table 7.8).

The patient with angina and another systemic disorder is best helped by a comprehensive approach taking into consideration the interaction between two diseases and the drug therapy. Especially important fact is that anemia often aggravates angina.

Angina is relieved by rest, sublingual nitroglycerine and sitting or standing (Table 7.9). In some patients angina is relieved by belching. Both sublingual nitroglycerine and sitting reduce venous return or preload. Additionally, nitroglycerine decreases aortic pressure (afterload). Relief with nitroglycerine should be prompt (2 minutes) for it to suggest the diagnosis of angina. Relief occurs with

Table 7.9: Angina pectoris: relieving factors

<i>Factor</i>	<i>Mechanism</i>
Rest	↓Heart rate ↓Blood pressure ↓MVO ₂
Sublingual nitroglycerine	↓Venous return (preload) ↓Blood pressure ↓MVO ₂
Sitting/standing	↓Venous return (preload) ↓MVO ₂
Belching	Reflex? Mechanism not clear
Walking through	Exercise induced peripheral vasodilatation Coronary vasodilatation? Recruitment of collaterals?

a mean time of 1.9 minutes with sublingual nitroglycerine but takes 2.9 minutes (chewable) and 3.4 minutes (sublingual) with isosorbide dinitrate (Sorbitrate). Relief occurring much later should suggest some other diagnosis. Relief with nitroglycerine may occur in esophageal spasm, gall bladder colic or any smooth muscle spasm. The tablets should be fresh, unexposed to light, should be chewed to powder and then kept under the moist tongue. In contrast to the exertional pain of classic angina pectoris, pain at rest is a feature of acute myocardial infarction and unstable angina. The pain of vasospastic angina also occurs at rest, usually at night.

The presence of rest pain means that there is absolute reduction in coronary flow and not relative increase in myocardial oxygen requirements (Table 7.10). The term mixed angina is applied when in a patient with exertional angina (or fixed angina due to fixed obstruction), occasional pain occurs at rest, particularly at night or early morning. This has therapeutic significance as a combination of betablockers and calcium blockers is effective.

Occasionally, in patient with 'mixed' angina, vasospastic angina may be precipitated by betablockers. Addition of calcium blockers relieves it. Angina induced by emotion usually lasts longer than exertional angina and may be mistaken for unstable angina. Nocturnal angina is generally suggestive of associated left ventricular failure but also occurs in a variety of other conditions.

Accompanying symptoms: The accompanying symptoms are those related to the abnormally contracting myocardium and the conducting tissue.

Table 7.10: Mechanisms of rest pain

<i>Cause</i>	<i>Mechanism</i>
Acute myocardial infarction	Occlusive thrombus superimposed over a preexisting coronary stenosis
Unstable angina	Atherosclerotic plaque rupture Thrombus Platelet aggregates
Vasospastic angina (Prinzmetal angina)	Coronary vasospasm
Mixed angina	Fixed obstruction with superimposed spasm
Emotion induced angina	Catechol excess Tachycardia Hypertension

a) Dyspnea: Shortness of breath occurs either as an anginal equivalent or as a manifestation of left ventricular failure. This symptom suggests a large area of myocardium at risk and these patients require early evaluation and revascularization. When shortness of breath is the presenting feature in a patient with no pulmonary disease, and normal left ventricular systolic function, one should consider the possibility of angina pectoris or diastolic dysfunction.

b) Syncope: Transient loss of consciousness may occur either due to the low cardiac output due to loss of contracting myocardium, or arrhythmia. Vasospastic angina or Prinzmetal angina may present with syncope of unexplained origin without chest pain. Though chest pain with syncope is often an indicator of coronary artery disease, alternative diagnostic possibilities should be considered (Table 7.11). Recurrent syncope due to chronic stable angina is distinctly uncommon and a single episode suggests an acute coronary syndrome, usually inferior wall myocardial infarction.

Due to the area of overlap, upper gastrointestinal disorders and acute pancreatitis are often mistaken for coronary artery disease.

c) Vomiting: Vomiting may accompany chest pain or may even be the presenting feature in myocardial infarction (Table 7.12). It is more likely to occur in the setting of acute inferior infarction due to excessive parasympathetic excess. Vomiting may also be the dominant feature in myocardial

Table 7.11: Causes of chest pain with syncope

<i>Condition</i>	<i>Mechanism</i>
Acute myocardial infarction	Large area of myocardium at risk Arrhythmias
Vasospastic angina	Arrhythmias (VT/VF)
Angina pectoris	Large area of myocardium at risk Arrhythmia
Acute pulmonary embolism	Obstruction to circulation
Pericardial tamponade	Cardiac compression
Tension pneumothorax	Cardiorespiratory embarrassment
Pleural hemorrhage	Cardiorespiratory embarrassment
Upper gastrointestinal bleeding	Loss of blood
Acute pancreatitis	Hypovolemia due to chemical peritonitis Severe pain

Table 7.12: Causes of vomiting in acute myocardial infarction

<i>Cause</i>	<i>Distinguishing feature</i>
Acute inferior MI	Other symptoms of parasympathetic excess like bradycardia
Drug induced	Morphine, aspirin, rarely nitrates
Cardiac rupture	Bradycardia, weakness, urge to pass stool Pericarditis type of pain

rupture occurring as a complication of acute myocardial infarction. Aspirin and morphine given in acute myocardial infarction may also be responsible for vomiting. Vomiting may also be a feature of acute right heart failure due to right ventricular infarction.

The esophagitis following vomiting often simulates the chest discomfort of acute coronary syndromes, and a mistaken diagnosis of coronary artery disease is often made.

d) Symptoms of autonomic excess: The symptoms related to excessive autonomic discharge are common in acute myocardial infarction and unstable angina but are uncommon with chronic stable angina (Table 7.13).

These symptoms of autonomic excess are suggestive of visceral origin to pain in contrast to somatic pain. They are not diagnostic of myocardial ischemia or infarction. In the absence of chest pain, one more of these symptoms may be the only indicators of myocardial ischemia or infarction.

Painless myocardial infarction or ischemia: A third of myocardial infarctions are unrecognized; these include completely asymptomatic (silent) events and those with atypical symptoms. The patient or the physician (when contacted) do not entertain the diagnosis of acute myocardial infarction.

Table 7.13: Symptoms and signs of autonomic excess

<i>Sympathetic excess</i>	<i>Parasympathetic excess</i>
Sweating	Weakness
Anxiety	Nausea
Fear	Vomiting
Palpitation (tachycardia)	Bradycardia
Hypertension	Hypotension

The causes of painless MI could be:

- In diabetic patients due to autonomic neuropathy
- In elderly patients with dementia or cerebrovascular disease
- Under general anesthesia
- In pulmonary edema when dyspnea is the dominant feature
- ? Betablocker therapy
- A quarter of patients do not have pain
- Failure on part of the patient/doctor to recognize the symptoms

Patients with diabetes are prone to silent myocardial infarction and silent exertional ischemia. Though the exact mechanism is unclear, it may reflect an impairment of the sensory innervation of the heart. Gamini et al studied anginal perceptual threshold in 32 diabetic patients and 36 non-diabetic control patients, all of whom had typical exertional angina. Anginal perceptual threshold was defined as the time from onset of 0.1 mV ST depression to the onset of chest pain during treadmill stress electrocardiography. Anginal perceptual threshold is delayed in the diabetic group compared to the controls.

When myocardial infarction is painless, the only clue can be the presence of one of the associated symptoms. Unexplained dyspnea, palpitation, syncope, or any of those symptoms listed above either in combination or in isolation should make one suspect myocardial infarction or ischemia. When myocardial infarction develops during anesthesia, unexplained hypotension, hypertension, tachycardia or bradycardia or ventricular arrhythmia may be the only clue. In a recent study Theisen et al observed that patients with unrecognised AMI showed greater 'alexithymia' or deficient psychological awareness and a greater belief that chance or fate determines their health. In India, religious beliefs, belief in alternative systems of medicine and economic factors also may play varying roles.

It is presently recognized that 60–80 per cent of ischemic episodes are asymptomatic. These silent ischemic episodes can be prolonged up to 40 minutes (Table 7.14).

Total ischemic burden is defined as the total duration of ischemia in minutes for 24 hours with or without symptoms. A total of more than 60 minutes connotes a poor prognosis. It has been proved that total ischemic burden is a better predictor of prognosis than symptoms alone.

Table 7.14: Classification of silent myocardial ischemia

<i>Type</i>	<i>Clinical subset</i>
Type I	Silent ischemia in asymptomatic patients (detected by exercise screening)
Type II	Asymptomatic post-myocardial infarction patients
Type III	In symptomatic stable or unstable angina

Symptoms due to associated disorders: In the management of patients with coronary artery disease, the presence of associated disorders has significant implications (Table 7.15).

Table 7.15: Significance of symptoms of associated disorders

<i>Condition</i>	<i>Significance</i>
Acid peptic disease	Difficulties in evaluation of pain Mistakes in diagnosis between acid peptic disease and clinical expressions of CAD are common Contraindication to thrombolytic therapy or anticoagulants and aspirin
Bronchial asthma	Mistakes in differential diagnosis with left ventricular failure (LVF) Betablockers contraindicated Allergic reactions to STK and contrast are more likely
Hypertension (> 110 mmHg, diastolic)	Contraindication to thrombolytic therapy Indication for aggressive management of hypertension during acute MI
Obstructive uropathy	The acute stress of MI, atropine, vasodilators and diuretics may precipitate acute retention of urine During interventions like PTCA, need for an indwelling Foley's catheter may arise
Recent cerebrovascular accident	May be a contraindication to thrombolytic therapy
Diabetes mellitus	Hypoglycemia may precipitate or simulate angina/left ventricular failure. Contrast induced renal failure is more likely
Renal failure	Choice and dosage of medication may need change Use of non-ionic contrast may be appropriate for angiographic procedures. Precautions need to be taken.

A thorough knowledge of the presenting manifestations of disorders producing pain in the chest is essential for a proper evaluation of each patient. A brief description of each of these entities is given below.

ANGINA PECTORIS

The typical characteristics of angina pectoris are shown in Table 7.16.

Table 7.16: Characteristics of angina pectoris

<i>Feature</i>	<i>Description</i>
Location	Substernal and a similar area over back Left shoulder and arm Interscapular area on either side Both shoulders Any where between upper abdomen to lower jaw Lower cervical or upper thoracic spine Lower jaw Only right shoulder or arm
Quality	Pressure like sensation or heaviness Constriction around chest or larynx or trachea Burning Tightness Shortness of breath Suffocating feeling Uneasiness Difficult to describe feeling Deep aching
Duration	0.5–30 minutes
Precipitating and relieving factors	Exercise Food Emotion Cold/hot environment Sexual intercourse Relief with nitroglycerine Relief with rest Relief with upright posture
Radiation	Medial aspect of left arm Left shoulder Jaw Occasionally right arm

EVALUATION OF CHEST PAIN: DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS

<i>Feature</i>	<i>Description</i>
Physical findings during angina	Heart rate and blood pressure Prominent <i>a</i> wave in JVP due to right ventricular ischemia Palpable precordial impulse due to dyskinesia S4 S3 in case of left ventricular failure (LVF) Mitral regurgitation due to papillary muscle dysfunction Crepitations over lung bases due to LVF.
ECG	Normal or ST segment depression or T wave inversion
Exercise testing	Exercise induced ST depression, fall in blood pressure, and wall motion abnormalities
Coronary arteriography	Fixed obstruction >75% in one vessel or >50% lesion in more than one vessel

Table 7.17: Functional classification of angina pectoris: (Canadian Cardiovascular Society + Goldman's specific activity scale)

Class I	Ordinary physical activity such as walking and climbing stairs does not cause angina. Angina with strenuous or rapid or prolonged exertion at work or recreation. Can perform any activity requiring 7 METs
Class II	Slight limitation of ordinary physical activity, walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals in cold in wind, or when under emotional stress or only during the few hours after awakening. Climbing more than one flight of ordinary stairs at a normal pace in normal conditions. Ordinary walking for less than two blocks on the level or climbing one flight of stairs does not cause angina. Can perform any activity requiring 5 METs, but cannot perform to completion any activity requiring 7 METs
Class III	Marked limitation of ordinary physical activity, walking less than two blocks on level ground and climbing one flight in normal conditions. Can perform any activity requiring 2 METs, but cannot perform to completion any activity requiring 5 METs
Class IV	Inability to carry on any activity without discomfort, anginal syndrome may be present at rest but must be brief (if longer than 15 minutes, it is called unstable angina) Cannot perform any activity requiring 2 METs

NOCTURNAL ANGINA

Chest pain awakening the person from sleep or occurring at night at rest is called nocturnal angina. The wider meaning of nocturnal chest pain extends to that of pain occurring at night. Nocturnal pain at rest implies sudden and severe decompensation of the supply and demand balance of myocardial perfusion. In the absence of occult and extreme increases in demand, such as accelerated hypertension or severe anemia, nocturnal pain refers to sudden loss of myocardial perfusion (that is, occlusive coronary artery disease) of any type.

The causes of nocturnal angina could be:

- Acute coronary syndromes
- Associated left ventricular failure
- Vasospastic angina
- Left main coronary artery disease
- Mixed angina
- Betablockers precipitating vasospasm
- Nocturnal angina with dreams due to propranolol
- Angina in severe aortic regurgitation
- Accelerated hypertension
- Inadequate spacing of drug therapy
- Nocturnal angina of sleep apnea

Vasospastic angina usually occurs at night or in the early hours of the morning since coronary vascular tone is highest at this time. In patients with mixed angina, the exertional angina responds to betablockers but the rest angina due to vasospasm continues or may get aggravated. This responds promptly to the addition of calcium blockers. Angina is less common in aortic regurgitation and often occurs at rest or nocturnally. The bradycardia of sleep increases aortic regurgitation, decreases the aortic diastolic pressure and increases the left ventricular diastolic pressure. As a result, the coronary perfusion pressure decreases, precipitating angina. The hypoxemia of sleep apnea may induce nocturnal angina. In the study by Franklin et al sleep apnea was present in 9 of 10 patients with nocturnal angina pectoris.

POSTPRANDIAL ANGINA

Postprandial angina is defined as angina occurring within 30 minutes of a meal

and repeatedly reproducible by the same stimulus. The common patterns are postprandial angina at rest, postprandial exertional, and isolated postprandial angina.

Postprandial angina pectoris is a well known symptom and was recognized by Heberden in 1772 and William Osler in 1897. The mechanism of postprandial angina is unclear. Postprandial redistribution of blood flow away from coronary circulation when it occurs at rest is likely. Redistribution to the gut and exercising muscles is likely in postprandial exertional angina. However, animal experiments in dogs and studies in human volunteers failed to support this idea. Other possible mechanisms suggested are coronary vasoconstriction secondary to postprandial gastric and esophageal dilation or gastric receptor stimulation, increased postprandial myocardial oxygen consumption (increased demand), increased postprandial cardiac output, postprandial lipid induced decrease in myocardial oxygen transport, and possible role of vasoactive intestinal peptide in modulating the cardiac response to food intake. A recent study by Berlinerblau et al suggested postprandial angina as a marker for severe coronary artery disease, and is often associated with unstable angina. It is often associated with left main or severe triple vessel disease.

UNSTABLE ANGINA

Unstable angina is defined as absence of ECG and enzymatic evidence of acute MI, crescendo angina on a preexisting chronic stable angina, angina at rest and on minimal exertion or angina pectoris of new onset (less than 1 month). It is also known as preinfarction angina, crescendo angina, acute coronary insufficiency or intermediate coronary syndrome.

As unstable angina comprises of a heterogeneous subset of conditions, the following classification is suggested by Braunwald. The severity, the clinical circumstance, and the presence or absence of ECG changes, and the therapy received and response to it were taken into consideration. The bases of the classification (Table 7.18) are the following factors:

- Severity of clinical manifestations
- Clinical circumstances
- Presence or absence of transient ECG changes
- Nature of therapy, and the response to it.

Table 7.18: Classification of unstable angina

Class I	Severity New onset, severe or accelerated angina Patients with angina of < 2 months Increase in severity, frequency, precipitated by lesser exertion. No rest pain in the last 2 months.
Class II	Angina at rest, subacute Patients with one or more episodes of angina at rest during the preceding month but not within the preceding 48 hours.
Class III	Angina at rest, acute Patients with one or more episodes at rest within the preceding 48 hours.
Class A	Clinical circumstances Secondary unstable angina A clearly defined condition extrinsic to the coronary vascular bed that has intensified myocardial ischemia. Anemia often due to gastrointestinal bleeding related to aspirin Fever Infection Hypotension Tachyarrhythmia Thyrotoxicosis Hypoxemia due to respiratory failure
Class B	Primary unstable angina
Class C	Postinfarction unstable angina < 2 weeks of myocardial infarction
Treatment	Intensity of treatment
1	No treatment or minimal treatment
2	Occurring in presence of standard therapy for chronic stable angina (conventional doses of oral betablockers or nitrates.)
3	Occurring despite maximally tolerated doses of all three categories of oral therapy, including intravenous nitroglycerine.

Unpredictability is the hallmark of unstable angina. It is important to recognize unstable angina because of the disabling nature of symptoms and the possibility of it ending in acute myocardial infarction.

The National Heart, Lung and Blood Institute published practice guidelines which defined unstable angina:

Unstable Angina is defined as having three possible presentations:

- Symptoms of angina at rest (usually prolonged > 20 minutes).

- New onset (< 2 months) exertional angina of at least Canadian Cardiovascular society Classification (CCSC) Class III in severity.
- Recent (< 2 months) acceleration of angina as reflected by an increase in severity by at least one CCSC Class to CCSC Class III.

In most, but not all, of these patients symptoms will be caused by significant coronary artery disease. Unstable angina has to be viewed as part of the spectrum of acute coronary syndrome. Variant angina, non-Q wave myocardial infarction, and post-MI (> 24 hours) angina are part of the spectrum of unstable angina.

ACUTE MYOCARDIAL INFARCTION

APPROACH TO A PATIENT WITH ACUTE CHEST PAIN

Both unstable angina and acute myocardial infarction require hospitalization and management. Before considering any interventions in acute coronary syndromes, rule out acute abdominal disorders simulating them. Pain in the chest is the commonest symptomatic expression of coronary artery disease (which is the commonest cause of death in adult population). A physician who is not proficient in evaluating it, is at a serious disadvantage in his practice and himself becomes an additional risk to the patient. Unfortunately, the present curricula of undergraduate and postgraduate education fosters this incompetence further.

Clinical features of acute myocardial infarction are given in Table 7.19.

Table 7.19: Coronary artery disease: acute myocardial infarction

<i>Feature</i>	<i>Description</i>
Precipitating factors	No precipitating factor in 50% of patients Heavy exertion Modest exertion Surgical procedure Sleep 8% Emotional stress 18% (increase in life change units) Within a few hours after heavy physical exercise Physical exertion under emotional stress or fatigue Drug withdrawal (betablockers, nitroglycerine in munitions workers) Ergot preparations as for migraine Respiratory infections Acute pulmonary embolism

CLINICAL METHODS IN CARDIOLOGY

<i>Feature</i>	<i>Description</i>
Time of the day (Circadian rhythm)	Hypoxemia due to any cause Hypoglycemia Vasospasm Tachycardia due to any cause Fever Early morning hours due to Catecholamines Cortisol Platelet aggregability (Exceptions to circadian periodicity Patients receiving aspirin and betablockers)
Prodromal symptoms	20–60% of patients have prodrome of angina 1–4 weeks prior to MI
Nature of pain at onset	Acute gradually increasing in severity, may wax and wane
Location/radiation	Retrosternal and a similar area over the back Spreads to either side of chest anteriorly more to left Ulnar aspect of the left arm with numbness in the wrist, hand and even fingers May radiate to shoulders, upper limbs, neck, jaw and interscapular region Rarely the pain may begin in the epigastrium
Duration	Prolonged > 30 minutes to many hours
Severity of pain	Variable (often severe, occasionally intolerable or mild)
Character	Heaviness, squeezing, choking, constricting, crushing, boring, stabbing, knifelike, burning, ‘palpitation’ In patients with past angina the pain is similar to angina but is longer and severe
Relieving factors	Usually not relieved by nitroglycerine May be relieved fully or partially if spasm is the dominant or associated feature Analgesics (morphine) may relieve it Thrombolytic agents may relieve it by recanalizing occluded vessel Congestive heart failure de novo or worsening of existing failure
Atypical presentations	Like classic angina pectoris Atypical location of pain Central nervous system manifestations Embolic stroke

EVALUATION OF CHEST PAIN: DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS

Feature	Description
Physical signs	<p>Transient ischemic attack due to low cardiac output with underlying cerebrovascular disease</p> <p>Apprehension and nervousness</p> <p>Psychiatric manifestations</p> <p>Syncope</p> <p>Extreme weakness</p> <p>Acute indigestion</p> <p>Peripheral embolism</p> <p>Tachycardia or bradycardia</p> <p>Hypertension or hypotension</p> <p>JVP elevated with CCF or RV infarction</p> <p>Palpable dyskinesia over precordium</p> <p>S4 or S3</p> <p>Mitral regurgitation</p> <p>VSD</p>
Angiographic correlation	Thrombotic occlusion of one of the major epicardial artery with a preexisting stenosis

Rapid identification and treatment of acute MI

If you cannot make a diagnosis, make a decision.

Chester M. Jones

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It is important to identify acute MI as early as possible (Table 7.20).

Table 7.20: Approach to a patient with acute chest pain: differential diagnosis

If non-cardiac, rule out	<p>Acute peptic ulcer syndromes</p> <p>Esophageal spasm/esophagitis</p> <p>Acute pancreatitis</p> <p>Acute cholecystitis</p> <p>Acute tension pneumothorax</p>
If cardiac, is it due to	<p>Coronary artery disease?</p> <p>Pericarditis?</p> <p>Acute pulmonary embolism?</p> <p>Aortic dissection?</p> <p>Mitral valve prolapse?</p> <p>Hypertrophic disorders?</p> <p>Severe aortic stenosis?</p> <p>Hypertrophic cardiomyopathy?</p>
If due to CAD is it due to	<p>Acute myocardial infarction?</p> <p>Unstable angina?</p> <p>Chronic stable angina?</p>

CLINICAL METHODS IN CARDIOLOGY

If due to chronic stable angina	Vasospastic angina?
	Mixed angina?
	What is the functional category?
	Is there occasional rest pain suggesting mixed angina?
If due to acute MI	Is there recent change in angina suggesting unstable angina?
	How many hours elapsed after the onset of pain?
	What is the extent of myocardium at risk?

Even if the diagnosis is uncertain, routine drug therapy (nitrates, betablockers, calcium blockers, antiplatelet agents) should not be delayed. The decision to discontinue the drugs can be taken after serial observation and follow-up stress test. Medical literature is full of information on how to handle patients with acute chest pain. Guidelines regarding admission and management into coronary care units are also provided based upon these studies Table 7.21 presents a possible approach to the management of such a patient.

One must realize that absence of proof is not proof of absence.

Table 7.21: Approach to management of a patient with acute chest pain

Category	Proposed action
Acute chest pain typical for acute ischemic syndromes with abnormal ECG	Do not waste time First things first. Check vital signs Insert IV line Monitor rhythm Bradycardia and hypotension: IV atropine/pacing Irregular pulse: monitor rhythm and appropriate treatment If the ECG shows evolving MI, make arrangements for thrombolysis or angioplasty.
Pain as above but no objective evidence	Manage as above but withhold thrombolysis Administer heparin, aspirin, nitroglycerine, nitrates, betablockers if no contraindication exists
Pain atypical but CAD can not be ruled out with confidence	Hospitalize but not necessarily in CCU / TRIAGE Unit Look for enzyme elevation including troponin and CPK-MB. Drug therapy as above but the physician should use judgment and individualize
Chest pain clearly non-cardiac	Rule out Acute peptic ulcer syndromes Esophageal spasm or esophagitis Acute pancreatitis Acute cholecystitis Acute tension pneumothorax Patient does not need hospitalization if the above conditions are ruled out

CHEST PAIN AND CORONARY ANGIOGRAPHY/ANGIOPLASTY

Chest pain is a common adverse event during diagnostic coronary arteriography. The incidence of severe angina is reduced by use of low osmolality contrast media. A recent study by Mathai et al showed angina related to type of contrast agent used but not to hemodynamic effects produced by the contrast.

The chest pain during coronary angioplasty is principally due to two mechanisms. One is related to myocardial ischemia during balloon inflation and the other related to mechanical stretch of the vessel wall itself. Fabrizio and associates showed that during coronary angioplasty, the cardiac pain experienced by patients is caused in part by stretching of the vessel wall. If the stretching is maintained at a constant level during repeated coronary occlusions, the cardiac pain is entirely predicted by the severity of myocardial ischemia and does not appear to be directly modulated by the mechanisms responsible for the ischemic preconditioning. Cardiac pain can be caused by chemical, thermal, and mechanical stimuli and the cardiac sensory receptors are polymodal. The presence of chest pain during angioplasty is an indication that the myocardium supplied by the vessel is viable.

The causes of chest pain due to coronary angioplasty are:

During angioplasty

- Myocardial ischemia
- Balloon inflation
- Mere presence of wire/balloon across a tight lesion
- Coronary spasm
- Damping of the main artery by guiding catheter
- Distal embolization
- Vessel wall stretch
- Pericardial pain due to perforation of vessel

Immediately following angioplasty

- Vessel closure
- Coronary spasm
- Non-cardiac pain

PERICARDITIS

The characteristics of pain due to pericarditis are given in Table 7.22. As pericarditis is usually due to some other systemic disorder, there is often evidence of systemic infection like tuberculosis, septicemia, malignancy, or myocardial infarction. It is important to recognize the associated pericarditis in acute myocardial infarction.

ACUTE PULMONARY EMBOLISM

The characteristics of the chest pain of acute pulmonary embolism are:

- Pleuritis type of pain in the axilla or interscapular region
- Central substernal pain like that of myocardial ischemia
- Dyspnea is the main symptom
- Central cyanosis may be present
- Sinus tachycardia is present
- Hypotension with elevated JVP and clear lungs

Sudden onset dyspnea in a patient with predisposing factors favours the diagnosis. Sinus tachycardia is always present and bradycardia rules out the diagnosis.

Table 7.22: Features of pericarditis

Site	Substernal, on either parasternal region Posterior or anterior cervical region Either trapezius or either shoulder, usually left Epigastrium or upper abdomen Radiation to arm or forearm is rare
Character and severity	Sharp or dull ache Pain may be very severe or mild Pain may be absent
Aggravating factors	Respiration Turning to side Lying on the back
Relieving factors	Sitting and leaning forward Steroids/analgesics
Associated features	Pericarditis is almost always secondary to some other systemic disorder or spread from other sites Pericardial rub Fever Signs of effusion/tamponade

Cyanosis though useful, is difficult to make out. The chest x-ray and ECG are often normal.

AORTIC DISSECTION

Acute aortic dissection though rare is often mistaken for the pain of acute myocardial infarction or unstable angina. Common features are:

- Sudden or dramatic onset of pain with gradual waning
- The pain is often ripping, tearing or stabbing
- Can simulate the pain of acute MI or unstable angina but is more commonly felt over the back
- Radiates to the neck, shoulders, abdomen or lower limbs if those arteries are involved in the process of dissection
- Features of acute myocardial infarction if the coronaries are involved in the dissecting process
- The murmur of AR if the aortic valve is involved
- Asymmetry or absence of arterial pulses
- Predisposing conditions like systemic hypertension, Marfan's syndrome

Aortic dissection can be confused for acute ischemic syndromes because of the similarity of pain and the frequently associated ST-T alterations of longstanding severe hypertension. It is important to differentiate between the two disorders because anticoagulant or thrombolytic therapy may be dangerous dissecting with

Table 7.23: Pain of aortic dissection: correlations

<i>Location of pain</i>	<i>Site of dissection</i>
Dominant anterior thoracic pain	Proximal dissection (DeBakey's Types I, II)
Dominant interscapular pain	Distal dissection (90%) (DeBakey's Type III)
Both anterior and posterior pain	Proximal and distal dissection
Absence of interscapular pain	Distal dissection is highly unlikely
Pain in the neck, jaw, teeth, throat	Dissection involving ascending aorta and arch

a hematoma of the aorta.

The location of pain may correlate with the site of dissection (Table 7.23). Aortic dissection should be 'looked for' in any patient with chest pain with any of the following presenting features (Table 7.24).

Table 7.24: Aortic dissection: possible presenting features

<i>Presenting feature</i>	<i>Mistaken for</i>	<i>Mechanism</i>
Acute chest pain	Acute MI Unstable angina	Aortic tear
Acute interscapular or low back pain	Musculoskeletal pain Acute disc prolapse	Aortic tear involving the descending thoracic or abdominal aorta
Asymmetry of arterial pulses	Embolic occlusion	Dissection involving branches of aorta
Aortic regurgitation (50% of proximal dissections)	Acute AR	Dilatation of aortic root and annulus by dissection Asymmetrical dissecting hematoma pressing on one of the leaflets Tear of annular support for leaflets or leaflets
Pericardial type of pain with rub or effusion or tamponade	Pericarditis Hemorrhaged cardiac tamponade (malignancy)	Dissection into pericardium
Neurological deficit	Acute stroke TIA Acute paraplegia Acute peripheral neuropathy	Proximal or distal dissection involving various arteries
Acute MI by ECG	Acute MI alone	Dissection involving coronaries
Acute abdominal pain/Ileus	Acute abdominal emergency	Dissection of abdominal aorta Mesenteric occlusion Renal infarction Renal failure
Severe hypertension, altered sensorium	Hypertensive encephalopathy	Renal failure
Fever	Fever of obscure origin	Resolving hematoma
Mediastinal widening	Mediastinal mass	Widened aortic shadow

In all patients presenting with acute chest pain, check for peripheral pulses to rule out dissection of aorta. When a patient appears to be in shock but has hypertension and has pain both above and below the umbilicus, aortic dissection should be suspected. Aggressive control of hypertension and appropriately timed surgery can be life saving. Unlike in acute ischemic syndromes, anticoagulants or thrombolytic agents are contraindicated in this condition.

MITRAL VALVE PROLAPSE

Mitral valve prolapse (MVP) is relatively common and may present with chest pain.

- Chest pain may suggest myocardial ischemia but is often atypical
- Patient is usually young, tall and lean
- Non-ejection click(s)/late systolic murmur on auscultation
- ECG shows ST, T alterations in inferior and lateral leads
- Exercise ECG may be falsely positive
- Echocardiography is diagnostic
- Carries a favourable prognosis in contrast to coronary artery disease.

Mitral valve prolapse should be distinguished from coronary artery disease. MVP generally carries an excellent prognosis in comparison to coronary artery disease. The mistake is compounded by the abnormal looking ECG, and false positive exercise test. Nitrates which are commonly used in coronary artery disease may aggravate the symptoms in mitral valve prolapse due to reduction in heart size.

NON-CARDIAC CAUSES OF CHEST PAIN

Esophageal pain

Pain of esophageal origin accounts for 10–25 per cent of acute chest pains admitted to emergency rooms to rule out acute myocardial infarction. Of all the causes of non-cardiac pain, esophageal pain simulates anginal pain most closely. The features of esophageal pain are listed below.

- Provocation by swallowing
- Oral regurgitation of liquid
- Retrosternal pain without lateral extension

- Relief by antacids
- Pain provoked by stooping
- Inconsistent relationship to exercise
- Frequent episodes of spontaneous pain
- Nocturnal pain
- Periods of prolonged remission
- Delayed response to nitroglycerine
- Accompanying symptoms (nausea, vomiting, swallowing difficulty, acid eructations)

The pain of esophageal origin shares some of the features of angina making the distinction difficult.

- Precipitated by exercise, emotion and food
- Pain radiating to left arm
- Relief by nitrates
- Nocturnal pain

Added to the above confusion, esophageal pain may precipitate myocardial ischemia in a patient with coexistent coronary artery disease. The association of the pain with swallowing, stooping forward, and oral regurgitation, and relief with antacids, will help in the differential diagnosis. These symptoms in patients less than 50 years favour an esophageal cause for the pain. As esophageal pain carries a benign prognosis, it should not be labelled angina. It is equally important to reach a positive diagnosis of esophageal pain before giving a benign prognosis as it is dangerous to miss the diagnosis of significant coronary artery disease.

When a woman presents with chronic chest pain, it is not enough to make a diagnosis of 'non-cardiac chest pain'. Rule out carcinoma of breast by asking for a mammographic examination. Delay in diagnosis of non-cardiovascular causes of chest pain may prove fatal in certain situations.

Case summary

A 45-year-old married lady presented with chest pain of 6 months duration. The pain was left sided and was variably related to exertion. The resting ECG and exercise ECG were normal. The echocardiogram was within normal limits. She had mild hypertension which was controlled with atenolol 50 mg per day. Her chest pain responded to alprazolam and antidepressants. She was seen by three cardiologists and one physician within a period of 9 months for hypertension and occasional chest discomfort. Nine months later, she had

fever with chills, redness and mild swelling of the left breast. A mammographic evaluation revealed a small discrete mass in the left breast. Aspiration cytology was suggestive of ductal carcinoma of breast with mild axillary extension. A radical mastectomy was done.

The term 'atypical chest pain' is often used when one or more features are atypical for angina. While symptoms may be due to myocardial ischemia, this description also includes chest pain with low probability of angina or non-cardiac chest pain. The term atypical chest pain does not rule out angina pectoris or myocardial infarction.

Chest pain of undetermined etiology

About 10–30 per cent of patients with chest pain suggestive of coronary artery disease are found to have normal or near normal coronaries. These patients were often labelled as having Prinzmetal angina (vasospastic angina), microvascular angina (small vessel disease), esophageal spasm or more commonly, syndrome X. The term syndrome X was used by Kemp in 1973, to denote the uncertainty of the etiology. Though there are differences of opinion about the pathophysiology of this syndrome, there is universal agreement that the long term survival of patients with chest pain syndromes associated with normal coronary arteries is excellent. The survival is not influenced by an ischemic response in ECG during exercise testing. The term microvascular angina was proposed by Cannon and Epstein in 1985, for this group of patients. These patients are suspected to have hypersensitivity of coronary microcirculation to vasoconstrictor stimuli with an associated limitation of microvascular vasodilator capacity. Dysfunction of the small intramural prearteriolar arteries may be site of abnormality. The demonstration of coexisting abnormal forearm hyperemic responses to ischemia, esophageal motility, and bronchoconstrictor responses to methacholine inhalation suggest a generalized disorder of vascular and other smooth muscle function. The demonstration of coronary microvascular dysfunction in patients with systemic hypertension who had chest pain, absent LVH and normal coronaries supports the concept. Some of these patients are shown to develop deterioration in left ventricular function in time. The common features in patients who have chest pain but have normal coronary vessels are:

- Hypersensitivity of coronary microcirculation to vasoconstrictor stimuli
- Limited vasodilator capacity

- Abnormal forearm hyperemic response to ischemia
- Esophageal motility disorder
- Bronchoconstrictor response to methacholine inhalation
- Ischemic exercise ECG response in some patients
- Deterioration of left ventricular function over time in some patients
- Exercise induced LBBB which over a period of time develops LBBB even at low heart rates.

In the last twenty years, investigation has not established any specific cause. As more information is obtained, the syndrome has become more confusing. For example, esophageal manometry when used in these patients sometimes revealed an abnormality, thereby converting a manometric abnormality into a 'disease'.

To many physicians and cardiologists, the absence of significant coronary artery disease in angiogram excludes the heart as a potential cause of pain. If these patients have relief with coronary vasodilators, it is important to continue the same. There are many case examples when these patients have developed acute myocardial infarction, when the coronary vasodilators were stopped abruptly. The patients with chest pain of psychological origin suffer as much or sometimes more than the patients with organic disease. Despite reassurance, many patients continue to have chest pain, receive drugs, frequent hospitalization, and diagnostic testing including cardiac catheterization. Adequate explanation and reassurance would be helpful. The patient's fears are best allayed by listening to him carefully and sympathetically.

EVALUATION AND MANAGEMENT OF PATIENTS WITH NON-ACUTE CHEST PAIN

In the management of patients with coronary artery disease, it is vital to estimate the severity of disease, for planning diagnostic studies and therapeutic approaches. A recent study by Pryor and his colleagues from Durham, North Carolina, clearly demonstrated the ability of simple history to predict the likelihood of severity of coronary artery disease. They studied the clinical characteristics of 6,435 consecutive patients that were most important in estimating the likelihood of severe disease. A list of these features is given below.

The ability of each characteristic to predict the likelihood of severity of disease increased with the presence of another characteristic. For example, in a patient

with typical chest pain, long duration of symptoms predicted severe disease more accurately. The presence of hypertension, diabetes, and smoking were more important predictors in women. Diabetes mellitus provides more prognostic information about women patients than any of the other traditional risk factors. In women presenting with chest pain, diabetes is the only risk factor that distinguishes those with angiographically verified coronary artery disease from those without it.

The **clinical characteristics** predicting severe CAD are:

- Age
- Gender
- Risk factors: Smoking, hypercholesterolemia, hypertension, diabetes mellitus
- Symptoms
- Pain type: Typical, atypical, non-anginal
- Duration of chest pain
- Pain frequency
- Accompanying dyspnea/syncope
- Evidence of myocardial damage
 - Congestive heart failure symptoms
 - ST-T wave changes
 - Ventricular gallop
 - Cardiac enlargement
- Premature ventricular complexes
- History of myocardial infarction
- Q waves on electrocardiogram
- Digitalis use
- Diuretic use
- Rales
- Heart murmur
- Left ventricular hypertrophy
- Conduction abnormalities
- Peripheral or cerebrovascular disease

The **determinants** of coronary artery disease in women with chest pain are:

Major

Typical angina pectoris

Postmenopausal status without hormone replacement

Diabetes mellitus

Peripheral vascular disease

Intermediate

Hypertension

Smoking

Lipoprotein abnormalities

HDL cholesterol levels

Table 7.25: Coronary artery disease: evaluation

<i>Symptom</i>	<i>Implication(s)</i>
Angina at rest within the preceding week.	<p>Absolute contraindication to exercise testing</p> <p>Calcium blockers should be given in addition to other antianginal agents, as vasospasm plays a dominant role in rest pain</p> <p>As the intracoronary thrombus is common in unstable angina, prior treatment with heparin and antiplatelet agents like abciximab, clopidogrel will facilitate better outcome.</p> <p>Direct coronary angiogram without exercise testing may be indicated and may be more cost-effective</p> <p>Surgical standby is needed even for coronary angiogram</p> <p>Facility to support the circulation by IABP is preferable</p>
Angina Class III or more	<p>Contraindication to exercise testing/ angiogram is indicated straightaway</p> <p>IABP/surgical standby is mandatory as the patient may start deteriorating after angiogram</p> <p>Non-ionic contrast is used preferably to avoid hemodynamic side effects of ionic contrast</p> <p>The number of injections should be limited</p> <p>The first few views in coronary angiogram should be directed to rule out left main coronary artery disease</p>
Chest pain associated with shortness of breath/syncope or hypotension	<p>Right heart catheterization is mandatory in all patients who present with dyspnea, to estimate pulmonary arterial wedge pressures</p>
History of TIA, carotid bruit	<p>Avoid the inadvertent entry of guide wire or catheter in neck vessels to avoid plaque disruption and embolism</p>

Minor

- Age > 65 years
- Obesity (especially central)
- Sedentary lifestyle
- Family history of coronary artery disease
- Other risk factors for CAD (psychosocial, hemostat)

Tables 7.25 and 7.26 describe the value of symptoms in planning diagnostic studies in patients with coronary artery disease and in making decisions in coronary artery disease after coronary arteriography.

Even a single episode of rest pain within the preceding week should be considered a contraindication to exercise testing. Exercise can precipitate serious arrhythmias or even acute myocardial infarction in this setting.

Table 7.26: Treatment implications after coronary arteriography

<i>Symptoms</i>	<i>Decision</i>
<i>Angina Class I</i>	Medical therapy is the choice
<i>Chronic stable angina</i>	No difference in survival between medical and surgical choices
<i>Class II</i>	
Mildly +ve or -ve stress test (Exercise capacity > 7 METS)	
<i>Angina Class III-IV</i>	
3 vessel disease with normal LV function	Excellent surgical outcome 5 years survival 92% with surgery Medical therapy 74% survival
3 vessel disease with LV dysfunction	Surgery definitely indicated Surgery 82% survival Medical 52% survival
Asymptomatic post-MI patient	CABGS does not prevent recurrent MI (CASS)
Left main obstruction > 50%, asymptomatic	Surgery indicated as sudden death is common without CABGS Surgery prolongs life
Asymptomatic patient with 50% lesion of one or more coronary arteries	Revascularization is not indicated as errors in interpretation are common in lesions of 40-70% The native disease progresses rapidly after surgery

PRACTICE IMPLICATIONS

- In a patient with acute chest pain, do not waste valuable time by waiting for an ECG to be done or going through all the details of family history. A 'first things first' approach is mandatory here. Check vital signs (pulse rate and blood pressure) even before asking for an ECG. If there is bradycardia and hypotension, intravenous atropine can be life saving.
- If the diagnosis of acute anterior myocardial infarction is obvious, start giving soluble aspirin and thrombolytic therapy without delay if no contraindication exists.
- If a patient has diffuse anterior chest pain for the first time in his life, take it seriously because it is almost always due to myocardial ischemia.
- When anyone comes to the emergency room at 3 AM in the night, always admit such a patient for observation even if you are clearly convinced that the pain is non-cardiac. This is for two reasons: one that the patient may not be telling you everything and you are not at your best at this time of the night. Always be impressed by the veracity of nocturnal symptoms such as headache, dyspnea, diarrhea, and chest pain – if they are serious enough to awaken the person from sleep, they constitute the most sincere physiological testimony and must be respected.
- A normal electrocardiogram in the presence of clinical syndrome of acute coronary ischemia does not rule out any acute ischemic syndrome or myocardial infarction. It is such a patient who can be helped most by the presently available methods.
- Continuing pain in acute myocardial infarction may mean continuing ischemia and calls for urgent intervention.
- It is unwise and dangerous to do an exercise test in a patient with history of rest anginal pain in the preceding week. Exercise test can be disastrous in this situation.

Case summary

A 60-year-old retired accountant consulted a cardiologist for exertional chest discomfort of 15 days duration and discomfort at rest of two days duration. The rest pain lasted 10–15 minutes and was typical of myocardial ischemia. He was known to have systemic hypertension for 20 years and diabetes mellitus for 15 years. The cardiologist did an exercise test during which the patient developed severe substernal discomfort along with

ECG changes suggestive of severe myocardial ischemia. The pain was partially relieved by sublingual nitroglycerine and he was advised to undergo coronary arteriography early and was sent home to take rest with a prescription of standard antianginal drugs and aspirin. The pain lasted for several hours but no electrocardiogram was done for next 2 days. Two days later he went to another hospital for recurrence of pain of 15 minutes duration and was hospitalized. The ECG showed anterior myocardial infarction of uncertain duration. After a 3 day course of intravenous heparin, he was subjected to coronary arteriography. This revealed total occlusion of left anterior descending and 70 per cent lesion of proximal right coronary artery. The left ventriculogram revealed severe hypokinesia of anterolateral wall with moderate dysfunction.

If your patient is asymptomatic but is advised bypass surgery or angioplasty ask for another opinion. The only exception is left main obstruction of more than 50 per cent.

Case summary

A 40-year-old businessman underwent a routine 'heart check' facilitated by a health card of medical insurance. Many tests were done including an echocardiogram and treadmill exercise test. The echocardiogram reported mitral valve prolapse and the computer printout of the treadmill test read 'Exercise test positive for myocardial ischemia; coronary arteriography must be considered'. As he was covered by insurance, coronary arteriography was performed and was interpreted as showing 50% narrowing of the right coronary artery and 50% narrowing of the circumflex artery with normal left ventricular function. He was referred to the cardiac surgeon who advised a prophylactic bypass operation. The patient who never had any symptom, without any clinical evaluation, found himself recovering from the operation, nursing a median sternotomy scar. He returned to work after 3 months but it took him another 3 months before he could resume his normal activities fully. The following year he underwent another routine check up. The treadmill test reported 'Exercise test strongly positive for myocardial ischemia; suggest coronary arteriography'.

This is a fictitious case summary, but could easily be real, as in some institutions, the overall philosophy exemplified is being followed. It is presented as an example of cardiology at its worst. Automation, gadgetry, assembly-line medicine and overreliance on laboratory findings may become substitutes for clinical judgment if the limitation of the methods we use is not realized (modified from Selzer).

- Evaluation and management of patients with chest pain is too important to be left to cardiologists and cardiac surgeons alone. The medical curricula should be modified to educate the medical student. Unfortunately, the Medical

Council of India which is the apex body formulating policies in medical education maintains that a medical student should not be exposed to the specialties during their student years. In real life, the level of general practice in any community is linked to proper education of medical students.

- ➔ The answer to the commercial practices of specialists lies in the general practitioner and practising physicians closely participating in decision making and management of their patients. They must update their knowledge from time to time by attending continuing medical education programmes and should work with an institution for a few weeks each year.
- ➔ If the cardiologist or cardiac surgeon points out a lesion in the angiogram, but you cannot see it, in all probability it is not present. Ask for another opinion.
- ➔ After a bypass surgery or angioplasty if the patient has pain similar to what was present before the procedure, it is always angina. The cardiac surgeons and cardiologists usually ascribe it to a musculoskeletal cause.
- ➔ It is a good practice to see the angiogram of your patient before decisions are made for them. This is the best way of learning to evaluate coronary angiograms.
- ➔ Learning to interpret a coronary angiogram is easier than interpreting chest pain. Most cardiologists and cardiovascular surgeons are only as intelligent as you are.
- ➔ If the patient's chest pain is atypical for myocardial ischemia, a positive exercise test does not necessarily establish that it is of ischemic origin. False positive exercise tests are common.
- ➔ The diagonal earlobe sign is completely useless in predicting the presence, absence or even severity of coronary artery disease. Many elderly individuals have it as an incidental sign.
- ➔ It is helpful to choose a working diagnosis and commit it to paper. Do not give a diagnosis as 'chest pain for evaluation'; specify 'myocardial infarction', 'unstable angina', or 'rule out unstable angina' or 'rule out acute myocardial infarction'. You do not need to be right, but you do need to allow for timely action. This serves to fix in the mind the signal observations, the criteria, for which the patient is being observed so closely and which will trigger decisive action. This is all the more important when the costs of inaction may exceed those of early action.

- When the electrocardiographic and clinical criteria suggest an evolving myocardial infarction of less than 12 hours duration, thrombolytic therapy should be given even if the chest pain is absent or subsides with nitroglycerine or morphine.
- Overreliance on stress thallium scintigraphy is a frequent source of error in decision making in coronary artery disease. It should not be used as a semifinal test before coronary angiography. When the chest discomfort is suggestive of myocardial ischemia, a negative thallium scintigraphy does not rule out the need for coronary arteriography if the patient has more than one risk factor for coronary artery disease. There is a common misconception among physicians and cardiologists that a negative thallium scintigraphy rules out the need for a coronary arteriography. This is wrong. The latitude with which thalliums are done and interpreted is highly variable from country to country and institution to institution. To rely on this test from the reported sensitivity and specificity of existing literature can be misleading.

Case summary

A 48-year-old physician consulted a cardiologist in November 1994 for palpitation and occasional episodes of chest discomfort variably related to exertion. He himself noted irregularity of pulse and was hospitalized for his arrhythmia. Earlier, in 1989, he was diagnosed to have angina pectoris and was receiving nitrates, aspirin, diltiazem and betablockers. An exercise ECG done at that time was positive for myocardial ischemia. He was a diabetic for 15 years with a family history of coronary artery disease. This time a stress thallium was done and was interpreted as negative for inducible ischemia. His symptoms and arrhythmia were considered related to overwork and exhaustion and he was advised to stop all medication.

Two months later, he woke up from sleep with severe anterior chest pain which was unrelieved by antacids. He was taken to a gastroenterologist and an upper GI endoscopy was done which was normal. As the pain continued even after 10 hours, the gastroenterologist felt that an ECG should be taken. The ECG revealed acute anterior myocardial infarction of several hours duration. As pain was continuing, 1.5 million units of intravenous urokinase was given. Coronary angiogram done before discharge showed a total occlusion of left anterior descending artery before the first septal with collaterals from the right coronary artery. Other coronaries were normal. Left ventricular systolic function was severely impaired and had an ejection fraction of 35 per cent.

In eight publications since 1983 involving over 4000 patients, the diagnostic sensitivity and specificity of thallium stress testing averaged 86 and 54 per cent

respectively. If corrected for referral bias, the specificity increases to 68 per cent and the sensitivity falls to 70 per cent. In symptom free individuals with risk factors, the diagnostic specificity is much lower, about 40 per cent or less.

LIMITATIONS OF CORONARY ANGIOGRAM

All symptomatic post-MI patients should directly have an angiogram. Non-invasive tests cannot help determine the need for angiogram.

It is widely believed that coronary arteriography is the gold standard test for the diagnosis of coronary artery disease. This is incorrect because coronary arteriography also has significant limitations.

Case summary

A 56-year-old executive was admitted in hospital with acute anterior chest pain typical of myocardial ischemia. The pain lasted for 20 minutes but the ECG, and serial myocardial enzymes were within normal limits. He was known to have systemic hypertension and diabetes mellitus for 5 years and never smoked. As the pain was typical, coronary arteriography was done and was interpreted as showing 25% lesion of proximal left anterior descending coronary artery. The other coronaries were normal with normal left ventricular function. He was told to ignore the symptoms and resume work. A week after discharge from the hospital, he sought another opinion with reconfirmation of the same findings. Fifteen days later, he was admitted with evolving anterior myocardial infarction which failed to respond to sublingual and intravenous nitroglycerine. He was given 1.5 million units of intravenous streptokinase with prompt relief of pain and resolution of ECG changes.

A repeat angiogram showed a 40% lesion in the proximal left anterior descending coronary artery with normal left ventricular function. This patient's story illustrates the limitations of coronary angiography.

A major limitation of the conventional angiographic assessment of coronary artery disease is that it disregards plaque morphology and it relies only on plaque stenosis. This purely anatomic-geometric approach is still standard practice despite evidence that coronary arteries are not passive conduits and a complex angiographic morphology is the marker of more profound endothelial damage. Plaque morphology is of recognized pathophysiological, clinical, and prognostic relevance but is ignored in clinical practice. Also currently it cannot be evaluated satisfactorily.

In all matters clinical, distrust everything and everybody, including yourself.

8 Dyspnea

Dyspnea is difficult and uncomfortable breathing. It occurs in a variety of situations. It is the commonest symptom of cardiac disease. Some of the common terms and their definitions are given below in Table 8.1.

Table 8.1: Definitions of terms

Dyspnea	Uncomfortable breathing
Tachypnea	Rapid breathing
Hyperpnea	Increased ventilation due to increased metabolic needs
Hyperventilation	Ventilation in excess of metabolic needs
Orthopnea	Dyspnea related to flat position
Platypnea	Dyspnea related to upright position
Trepopnea	Dyspnea related to lateral position

The **causes** of dyspnea are

- Cardiac
- Pulmonary
- Anemia
- Obesity
- Hysterical/psychogenic
- Malingering
- Physical deconditioning

In practice, cardiac and pulmonary causes are the most common. Pulmonary venous hypertension (PVH) is the basis for dyspnea in most cardiac disorders. In pulmonary venous hypertension, transudation of fluid from intravascular to extravascular space occurs when the pressures in the capillaries exceed 25 mmHg.

This results in greater stiffness of the lungs, increase in the work of breathing and increased resistance to airflow. The ventilator drive is increased due to stimulation of stretch receptors in lung vessels and interstitium. The hypoxemia and metabolic acidosis increase the ventilator drive.

The **mechanisms** of cardiac dyspnea are

- Increased mechanical work of breathing
 - Decreased compliance of lungs
 - Increased blood volume in the lungs
 - Increased vascular pressure in the lungs
 - Decreased air volume
 - Interstitial edema in the lungs
 - Increased airway resistance
 - Bronchiolar compression
 - Airway edema
- Increased ventilator drive
 - Hypoxemia
 - Ventilation-perfusion mismatch
 - Acidosis
 - Increased PaCO_2
 - Increased lactic acid
- Fatigue of respiratory muscles
- Decreased cardiac output

EVALUATION

For proper management of the patient with dyspnea, the following checklist of questions is useful in practice.

- Is it dyspnea, or a condition simulating it?
- If dyspnea, is it of cardiac or pulmonary cause?
- If cardiac, what is the grade of dyspnea?
- Is there paroxysmal nocturnal dyspnea and orthopnea?
- What is the duration of the symptoms?
- What is the time interval between the onset of dyspnea and edema?
- Is the patient receiving drugs; if so what is the response?
- What are the associated symptoms?

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Some patients with coronary artery disease present with 'shortness of breath' instead of chest discomfort. This should be interpreted as an anginal equivalent. In contrast to true dyspnea, these patients on careful questioning admit to the fact that they have to stop walking for a while for relief of dyspnea. If a 50-year-old patient with a background of diabetes, hypertension and smoking presents for the first time in life with 'dyspnea' with a normal cardio-respiratory system, angina pectoris should be ruled out by proper testing. Depression with sighing and fatigue is common and may be mistaken for dyspnea.

The rapid and deep breathing of metabolic acidosis is recognized by lack of sustaining evidence of left ventricular failure and alteration in the sensorium that often accompanies metabolic acidosis. Even slight drowsiness is an important clue because left ventricular failure does not produce alteration in sensorium unless there is severe hypoxia or hypotension. Most patients with acute pulmonary edema are anxious and alert. Drowsiness supervenes only when they are severely hypoxic or hypotensive.

Conditions simulating dyspnea are

- Dyspnea as anginal equivalent
- Acidotic breathing (Kussmaul's breathing): Diabetic ketoacidosis, renal failure
- Hyperventilation
- Anxiety/hysteria
- Malingering
- Acute pulmonary embolism
- Central neurogenic hyperventilation
- Drug induced dyspnea: Salicylates, methyl xanthine derivatives, beta adrenergic agonists, progesterone
- Pregnancy
- Fever
- Septicemia
- Depressive illness
- Sleep apnea
- Protracted cough

Drowsiness in left ventricular failure has diagnostic implications (discussed above) as well as therapeutic significance. Traditionally, patients with left ventricular

failure and pulmonary edema are given morphine to alleviate the anxiety and reduce the work of breathing. Though morphine is the 'drug of choice' in this setting, in the special subset of patients with pulmonary edema and drowsiness, morphine depresses the respiratory centre further and induces respiratory arrest. In this situation, morphine should be given only when the facility for mechanical ventilation is readily available.

The causes of dyspnea with drowsiness are:

With the rapid deep breathing (Kussmaul's) of metabolic acidosis

Renal failure

Diabetic ketoacidosis

Methyl alcohol poisoning

With dyspnea of left ventricular failure

Associated metabolic encephalopathy

Hypoglycemia

Diabetic ketoacidosis

Renal failure

Hyponatremia

Associated severe hypoxia/hypotension preterminally

Drug induced (morphine)

With central nervous system involvement

Central neurogenic hyperventilation

Hypertensive encephalopathy

Central neurogenic pulmonary edema

Embolic stroke

Aspiration pneumonia in any unconscious patient

Occasionally the condition responsible for drowsiness may trigger left ventricular failure and the causative disorder may go unnoticed. The following case summary illustrates this.

Case summary

A 68-year-old male was admitted to the emergency ward with acute pulmonary edema. He was known to have hypertension, diabetes and coronary artery disease, anterior myocardial infarction with left ventricular dysfunction. He was on therapy with nifedipine, enalapril, oral nitrates, and oral hypoglycemic agents. At admission, pulse rate was 124/minute, regular, BP 180/110 mmHg. Lungs showed bilateral coarse rales consistent

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with pulmonary edema. Auscultation of the heart was unrewarding due to noisy respiratory sounds. He was given intravenous furosemide and intravenous nitroglycerine with mild relief of pulmonary edema. Samples were sent for routine biochemical investigations but the reports were not immediately received. As the patient was found to be drowsy, hypoglycemia or cerebrovascular accident were considered. The blood sugar was 28 mg%. He was given intravenous glucose and recovered promptly.

Drowsiness often goes unobserved in many clinical settings. Even when noticed, it is explained away as being drug induced. The adrenergic excess of hypoglycemia can precipitate left ventricular failure by aggravating myocardial ischemia and systemic hypertension. The symptoms of left ventricular failure may precede or dominate the symptoms of neuroglycopenia (altered sensorium).

Once dyspnea is distinguished from conditions simulating it, differentiate whether it is cardiac or pulmonary.

Features in dyspnea suggesting pulmonary cause:

- Cough with expectoration
- Wheezing
- May not be related to exertion
- Fever
- Pleural type of pain
- Loss of weight
- Seasonal variation
- Progressive increase over many years
- Prompt response to bronchodilators
- Prompt response to oxygen
- Deep cyanosis in adults
- Absence of heart disease by physical examination or investigation.

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In elderly patients with systemic hypertension, known to have coronary artery disease and chronic obstructive pulmonary disease it may be difficult to pinpoint the cause.

Features in dyspnea suggesting cardiac cause:

- Paroxysmal nocturnal dyspnea and orthopnea
- Associated symptoms of heart disease
- Expectoration of brown, frothy sputum

Rapid progression of symptom
 Little or no cyanosis with severe dyspnea
 Response to diuretics and digoxin
 Evidence of heart disease by physical examination and investigation
 Cheyne–Stokes breathing

In some situations it is difficult to distinguish between cardiac and pulmonary dyspnea on the basis of the clinical examination. Investigations like ECG, X-ray, echocardiogram, pulmonary function tests and arterial blood gases may be needed.

DURATION AND ONSET OF SYMPTOMS

It is important to know the total duration and the time of onset of symptoms of right heart failure. If the duration of dyspnea is longer than 5 years particularly with paroxysmal nocturnal dyspnea and orthopnea, mitral stenosis is the most likely diagnosis. Other disorders like aortic valve disease and coronary artery disease are less likely as most of these patients do not survive longer than 3 years after the onset of heart failure. The time interval between dyspnea and symptoms of right ventricular failure is longer in mitral stenosis than in the other causes of left heart failure. In mitral stenosis the right ventricle fails under high pressures of pulmonary artery. This is because it hypertrophies due to the slowly developing pulmonary arterial hypertension in mitral stenosis. In other disorders like aortic valve disease or myocardial infarction, the pulmonary venous and arterial pressures are suddenly elevated with the development of left ventricular failure on a right ventricle which was unprepared by prior hypertrophy. In chronic aortic regurgitation, if the duration of dyspnea is more than 18 months, the outcome of surgery is poor.

MEASUREMENT OR GRADING OF DYSPNEA

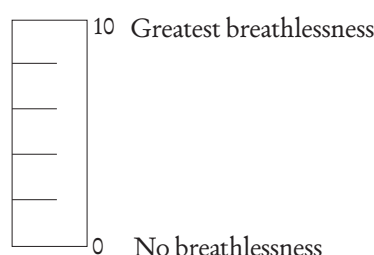
The degree of disability by dyspnea can be graded semi-objectively. In dyspnea due to pulmonary venous hypertension (PVH), the degree of dyspnea correlates with severity of pulmonary venous hypertension.

The visual analogue scale (VAS) can also be used for grading dyspnea. The VAS is a vertical line of 100 mm with the bottom labelled 'no breathlessness' and the top 'greatest breathlessness'. Similarly, the Borg category scale can be used which ranges from 0–10.

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Table 8.2: Grading for dyspnea

<i>Grade</i>	<i>Degree of exertion</i>	<i>PVP (mmHg) at rest</i>
Grade 1	Severe unaccustomed exercise	< 12 (normal)
Grade 2	Moderate exertion	12–18
Grade 3	Mild exertion	19–24
Grade 4	Dyspnea at rest	>25



VISUAL ANALOGUE SCALE (VAS)

A patient may be shown this chart and explained that greatest breathlessness corresponds to 10 and no breathlessness to 0. Then he may be asked to point out to where his breathlessness would fit in. This will be a useful way of assessing his breathlessness.

A patient with paroxysmal nocturnal dyspnea is graded as having grade 3 dyspnea even if the patient is not dyspneic on mild exertion. Orthopnea is obviously ranked as grade 4. Whatever the grade, drug therapy with diuretics and digoxin should be mentioned. The presence of paroxysmal nocturnal dyspnea and orthopnea strongly suggest pulmonary venous hypertension as the underlying cause of dyspnea.

Paroxysmal nocturnal dyspnea (PND)

The patient wakes up 2–3 hours after going to sleep with shortness of breath and cough, sits up on the bed or stands and opens the window as if hungry for air. Usually the dyspnea subsides spontaneously but sometimes may progress to frank pulmonary edema. Paroxysmal nocturnal dyspnea is related to interstitial pulmonary edema. The most important mechanism of paroxysmal nocturnal dyspnea appears to be absorption of edema fluid from the interstitial compartments of the lower limbs in a supine position with increase in venous return to the right heart. When the right ventricular output exceeds that of the left ventricular emptying, pulmonary

edema occurs. More than one of the following mechanisms may be responsible:

- Absorption of edema fluid with increase in right ventricular output overfilling the lungs
- Diminished sympathetic drive of sleep decreasing LV contractility
- Sleep induced dreams with attendant increase in emotional activity
- Nocturnal arrhythmias
- Sleep apnea (central neurogenic hypoventilation)

Once the right ventricle fails, paroxysmal nocturnal dyspnea usually disappears and gives way to easy fatiguability, a reflection of low cardiac output. Paroxysmal nocturnal dyspnea is not always diagnostic of left heart failure as nocturnal episodes of dyspnea occur in a variety of conditions.

Conditions simulating PND are:

- Nocturnal episodes of asthma
- Nocturnal episodes of recurrent minute pulmonary emboli
- Sleep apnea with arousal
- Cheyne-Stokes respiration
- Postnasal discharge with attendant severe cough
- Anxiety with hyperventilation
- Nocturnal angina with dyspnea as anginal equivalent
- Obesity
- Nocturnal aspiration in gastroesophageal reflux disease.

The patient with bronchial asthma is younger, with a history of similar illness earlier in life. Recurrent episodes of dyspnea with clear lungs in any patient with unexplained pulmonary arterial hypertension, should suggest the possibility of recurrent pulmonary emboli.

Orthopnea

This refers to increase in dyspnea in supine posture and relief by sitting up or upright posture. This is related to increased venous return in supine position with increase in right ventricular output, further increasing the pulmonary venous congestion. Though orthopnea is generally suggestive of left heart failure, it may also occur in chronic obstructive lung disease or any condition with large ascites encroaching on the lung volume. The causes could be:

- Left heart failure

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- Chronic obstructive pulmonary disease
- Any condition with significant ascites
- Constrictive pericarditis
- Any severe right ventricular failure
- Bilateral diaphragmatic paralysis

There is a mechanical advantage to breathing in the upright posture compared to the supine position. For this reason, in all situations with respiratory distress, many patients feel more comfortable sitting up.

Table 8.3: Drugs aggravating or relieving dyspnea

<i>Betablockers</i>	
Aggravate	Bronchospasm Ventricular failure Depression
Relieve	Anxiety Angina pectoris (anginal equivalent)
<i>Bronchodilators</i>	
Aggravate	Anxiety (salbutamol) Arrhythmia related dyspnea Mitral stenosis (tachycardia) Angina pectoris (tachycardia)
Relieve	Bronchospasm
<i>Nitrates</i>	
Aggravate	Angina of HOCM, MVP (anginal equivalent)
Relieve	Ventricular failure
<i>Digoxin</i>	
Aggravate	Angina or dyspnea in HOCM
Relieve	Mitral stenosis with atrial fibrillation Ventricular failure
<i>Diuretics</i>	
Aggravate	Pericardial disease Obstructive pulmonary disease Diastolic dysfunction
Relieve	Left heart failure Renal failure
<i>Steroids</i>	
Aggravate	Heart failure Renal failure
Relieve	Bronchospasm

Response to drugs: Dyspnea should be graded keeping the drug therapy in mind, as drugs like diuretics significantly influence pulmonary venous pressure and may even normalize it (Table 8.3). Grading of dyspnea has a direct bearing on functional categorization by NYHA. The patient's prognosis may be wrongly categorized if the influence of drug therapy is not considered. Almost all the studies on valvular heart disease use NYHA functional classification. Both relief and aggravation of dyspnea give valuable clues to the underlying mechanisms or causes of dyspnea.

As drug therapy can widely influence many disorders, careful drug history and the sequence of progression or regression of symptoms give useful clues.

CHEYNE-STOKES BREATHING

The chemoreceptors controlling ventilation are effected by oxygen and carbon dioxide tensions in the blood. In a normal circulation, the transfer of information is prompt with a normal circulation time. In heart failure and in cerebrovascular disease, prolonged circulation time and abnormal neural control can produce Cheyne–Stokes breathing.

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The various mechanisms are:

- Heart failure
- Prolonged circulation time
- $\uparrow\text{CO}_2$ sensitivity due to pulmonary congestion
- $\uparrow\text{CO}_2$ sensitivity due to arterial hypoxemia
- Decreased O_2 and CO_2 storage in lungs
- Associated cerebrovascular disease
- Neurologic disease
 - $\uparrow\text{CO}_2$ sensitivity (cortical/brainstem disease)
 - $\downarrow\text{CO}_2$ sensitivity with O_2 dependence
 - $\uparrow\text{CO}_2$ threshold
 - Loss of wakefulness drive
 - \downarrow Cerebral blood flow response due to changes in CO_2

If cerebrovascular accident is excluded, Cheyne–Stokes breathing is diagnostic of left ventricular failure.

DYSYPNEA IN VARIOUS CLINICAL STATES

CORONARY ARTERY DISEASE

Dysypnea is often an anginal equivalent implying that all patients with unexplained dysypnea require evaluation for coronary artery disease. Dysypnea in association with chest discomfort usually means a significant area of myocardium is at risk, and is an indication for more aggressive interventions in the acute coronary syndromes.

Left ventricular failure in myocardial infarction generally means a loss of 25 per cent or more of myocardium and the ejection fraction is usually less than 40 per cent. Though other signs of left ventricular failure like third heart sound are helpful, dysypnea is the most sensitive feature. If the patient has dysypnea with ejection fraction of more than 40 per cent, conditions listed below should be suspected.

- Anginal equivalent
- Left ventricular failure due to
 - Ischemia
 - Infarction
 - Ventricular aneurysm
 - Mitral regurgitation
 - Ventricular septal defect
 - Arrhythmias
 - Pulmonary embolism
- Betablocker induced bronchospasm
- Associated chronic obstructive airway disease
- Extreme weakness of physical deconditioning due to prolonged bed rest
- Associated metabolic abnormality either simulating or aggravating left ventricular failure
- Renal failure
- Diabetic ketoacidosis
- Lactic acidosis

Dysypnea is more common in anterior myocardial infarction than in inferior myocardial infarction because of the larger area of myocardium involved in the

former. Dyspnea in a patient with inferior myocardial infarction means an associated lateral or true posterior myocardial infarction, mitral regurgitation, ventricular septal defect, old anterior myocardial infarction, associated ischemia of anterior wall, or some other disorder. In patients with diabetes mellitus the site and size of infarction on the ECG may not correlate with the degree of left ventricular dysfunction.

The causes of dyspnea in inferior myocardial infarction are:

- Inferior myocardial infarction + lateral myocardial infarction/true posterior myocardial infarction
- Old anterior myocardial infarction
- Ischemia of left coronary territory
- Mitral regurgitation
- Ventricular septal defect
- Inferior myocardial infarction in a diabetic patient
- Pulmonary embolism
- Pulmonary embolism mistaken for inferior myocardial infarction
- Diabetes mellitus with cardiomyopathy

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Once dyspnea of heart failure occurs after myocardial infarction, the 4 year survival rate is only 50 per cent in contrast to patients who have 80 per cent survival without heart failure and more than 50 per cent ejection fraction (CASS study).

VALVULAR HEART DISEASE

Mitral stenosis

Dyspnea is the initial and most important symptom of mitral stenosis in contrast to other lesions where it is a late manifestation. Dyspnea occurs from the beginning of the disease because pulmonary venous hypertension is an intrinsic part of any significant mitral valve obstruction. This is unlike aortic valve disease where pulmonary venous hypertension occurs late in the disease with the onset of left ventricular failure. Unlike aortic valve disease, patients with mitral stenosis live beyond 5 years after the onset of dyspnea. Once dyspnea occurs in aortic valve disease, survival beyond 3 years is unusual since once left ventricular dysfunction occurs, there is rapid deterioration. As a general rule, if a patient has dyspnea with paroxysmal nocturnal dyspnea for more than 5 years, mitral stenosis is most likely

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to be present and aortic valve disease is unlikely. Dyspnea in mitral stenosis is related to alterations in lung function secondary to pulmonary venous hypertension (PVH). As the severity of pulmonary venous hypertension increases, all aspects of lung function deteriorate.

The alterations in lung function that take place in mitral stenosis are:

- Decreased vital capacity
- Decreased forced expiratory flow rates and volumes
- Decreased maximum breathing capacity
- Decreased dynamic compliance
- Decreased arterial oxygen tension
- Increased airway resistance
- Increased alveolar-arterial gradient for oxygen

These features correlate well with the increase in pulmonary arterial pressure and vascular resistance. Features of right heart failure appear many years after the onset of symptoms of left heart failure in mitral stenosis. The hypertrophied right ventricle is capable of working despite pulmonary arterial hypertension for many years. In lesions beyond the mitral valve, symptoms of right heart failure closely follow that of left heart failure as the non-hypertrophied right ventricle is not capable of working under significant pressure overload and fails at lower pulmonary arterial pressures. After the first episode of rheumatic fever, there is always a latent period of at least 3 years before dyspnea appears in mitral stenosis as mitral stenosis takes time to develop.

The severity of dyspnea has prognostic implications in mitral stenosis (Table 8.4). In mitral stenosis, it takes 3–5 years for dyspnea to progress from functional class II to functional class IV. Only 15 per cent of patients of class IV survive 5 years after the diagnosis. In the absence of significant dyspnea, paroxysmal

Table 8.4: Degree of dyspnea: effect on survival in mitral stenosis

<i>NYHA functional class</i>	<i>10 years survival</i>
Class I	85%
Class II	50%
Class III	20%
Class IV	None

nocturnal dyspnea or orthopnea, if a patient with mitral stenosis has features of right heart failure, associated tricuspid stenosis should be suspected. Only 2 per cent of patients with severe mitral stenosis and pulmonary arterial hypertension present with right ventricular failure straightaway without ever having had severe dyspnea or paroxysmal nocturnal dyspnea.

Aortic stenosis

Angina, syncope and dyspnea are the three cardinal symptoms of aortic stenosis. Dyspnea is the most menacing of the three symptoms. The average survival after the onset of dyspnea is only 1.5 years but is 2–3 years with syncope and angina. If the duration of dyspnea is longer than 5 years in aortic stenosis, an associated mitral valve disease should be suspected. Dyspnea in a patient with ‘mild aortic stenosis’ may suggest underlying mitral valve disease, coronary artery disease or hypertrophic cardiomyopathy. In hypertrophic cardiomyopathy, the symptoms have no correlation to the presence, absence or the degree of outflow obstruction. The fundamental mechanism of dyspnea in hypertrophic cardiomyopathy is diastolic dysfunction related to the restriction to ventricular filling leading to pulmonary venous hypertension.

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The causes of dyspnea in mild aortic stenosis are:

- Hypertrophic cardiomyopathy
- Coronary artery disease
- Associated mitral valve disease
- Unrelated disorder (pulmonary)

Though dyspnea is often a symptomatic expression of left ventricular systolic dysfunction in aortic stenosis, it may be due to diastolic dysfunction of severe left ventricular hypertrophy. The exertional angina of aortic stenosis may be mistaken for dyspnea.

Mitral regurgitation

Palpitation is usually the first symptom in mitral regurgitation and dyspnea follows it. Dyspnea in mitral regurgitation is usually due to left ventricular failure, but may also be due to elevated left atrial pressures related to severe mitral regurgitation into a non-dilated, non-compliant left atrium. Unlike mitral stenosis, dyspnea is a later symptom in mitral regurgitation. Tachycardia increases the frequency of *v* waves in left atrium and pulmonary veins increasing the mean pulmonary venous

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pressure. The mechanisms of pulmonary venous hypertension in MR are:

- Left ventricular failure
- Severe mitral regurgitation with non-compliant LA
- Tachycardia (more *v* waves)
- Associated MS

Sudden onset of dyspnea in a patient with mitral regurgitation may suggest recurrence of rheumatic activity, infective endocarditis, chordal rupture or the onset of atrial fibrillation. The causes of rapid progression of dyspnea in mitral regurgitation are:

- Infective endocarditis
- Recurrence of rheumatic activity
- Chordal rupture
- Onset of atrial fibrillation
- Onset or progression of coronary artery disease

Appropriate timing of surgery for mitral regurgitation is important and most of the presently available investigations have serious limitations. The onset of dyspnea is the most important event that suggests the beginnings of ventricular dysfunction. Dyspnea of NYHA functional class II is presently considered an indication for surgery. The duration of dyspnea is important because if the symptom has been present for less than one year the surgical outcome is good. On the other hand, if the duration of dyspnea is more than 2 years, significant deterioration in left ventricle function is likely with a poor surgical outcome. If the duration of dyspnea is more than 5 years with obvious biventricular failure, many of these patients have poor left ventricular function and are at a very high risk for surgery. Evaluation must be based on:

- Onset
- Grade/functional class
- Duration of the symptom
- Diuretics/digoxin

As the onset of dyspnea triggers the decision for surgery in mitral regurgitation, it is important not to give diuretics and digoxin to these patients with minimal or no symptoms. When a diuretic or digoxin is given, the decision for surgery is already made. As diuretics and digoxin mask the beginnings of dyspnea, advanced

left ventricular dysfunction may exist without being symptomatic. Objective testing of functional class and serial echocardiographic evaluations are helpful.

Aortic regurgitation

Dyspnea occurs very late in the course of aortic regurgitation and is suggestive of left ventricular failure. If dyspnea occurs early in the clinical course of aortic regurgitation, one must consider associated mitral valve disease or acute aortic regurgitation, acute on chronic aortic regurgitation or associated diseases. The symptom is not only late to appear but is slow in progressing. Rapid progression of dyspnea in aortic regurgitation should elicit the possibility of infective endocarditis, retroversion of the aortic cusp, or the onset of systemic hypertension.

The causes could be:

- Recurrence of rheumatic activity
- Infective endocarditis
- Retroversion of the aortic cusp
- Aortic dissection
- Onset of systemic hypertension.
- Associated coronary artery disease

Dyspnea of class II, III or IV should be considered an indication for surgery in aortic regurgitation if other causes of dyspnea are ruled out. Left ventricular dysfunction may exist in aortic regurgitation without dyspnea due to a compliant left ventricle. If the duration of dyspnea is less than 2 years, the outcome of surgery is good. Some patients with aortic regurgitation, feel better with walking than at rest. This is related to exertional increase in heart rate decreasing the amount of aortic regurgitation, and fall in peripheral vascular resistance, which also reduces the degree of aortic regurgitation. However, once left ventricular failure occurs, exertion always aggravates dyspnea.

PULMONARY EMBOLISM

Dyspnea is the most common and consistent symptom of acute pulmonary embolism. Usually it is of sudden onset with acute presentation. However, recurrent pulmonary emboli can result in pulmonary arterial hypertension which may cause exertional dyspnea. The severity of the symptoms depend upon the extent of the pulmonary arterial tree involved.

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The electrocardiographic patterns of pulmonary embolism may be mistaken for inferior myocardial infarction plus true posterior myocardial infarction due to the Q waves in L III, and taller R in V1.

RIGHT HEART FAILURE

Though significant dyspnea is generally considered a feature of left heart failure, right heart failure also produces dyspnea. Gibbs et al clearly showed the lack of correlation between pulmonary artery (capillary) pressure and dyspnea. It is now generally accepted that the mechanism of breathlessness is too complex to be explained by alterations in pulmonary capillary pressures alone. Respiratory muscle dysfunction plays an important role. Sullivan et al demonstrated that an abnormal increase in physiologic dead space per breath is the primary mechanism of breathlessness in heart failure. They hypothesized that ventilation-perfusion mismatch due to an abnormally low cardiac output is responsible. As right ventricular dysfunction reduces the pulmonary perfusion, the pulmonary arterial pressures or (indirectly the capillary pressures) fall. In any case, right ventricular dysfunction appears to be an important determinant of dyspnea but fails to produce paroxysmal nocturnal dyspnea or orthopnea.

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ARTERIAL BLOOD GAS (ABG) ANALYSIS

In present day critical care practice it is essential to know how to interpret arterial blood gases. Arterial blood gas analysis not only aids in diagnosis, but is also useful in guiding subsequent therapy. Normal values are given below:

pH:	7.38–7.44
PO ₂ :	80–100 mmHg
PCO ₂ :	35–45 mmHg
HCO ₃ ⁻ :	21–28 meq/l
SaO ₂ :	97–100%

Interpretation of arterial blood gases is extremely useful in the evaluation of unexplained acute dyspnea. The common patterns in various conditions are given in Table 8.5.

At times the interpretation may be compounded by a combination of disorders.

Table 8.5: ABG analysis in various disorders with dyspnea

	<i>pH</i>	<i>PO₂</i>	<i>PCO₂</i>	<i>HCO₃</i>	<i>RR</i>
Acute pulmonary edema	→↓	↓ to ↓↓↓	→↓	↓ to ↓↓↓	↑↑
Cardiogenic shock	↓↓↓	↓↓	↓↓	↓↓↓	↑↑
Acute pulmonary embolism	→↓	↓↓	↓↓	→↓	↑↑
Cardiac shunt (R-L)	→↓	↓↓	→	→	→↑
Hypoventilation					
neural	↓	↓↓	↑↑↑	→↑	↓
neuromuscular	↓	↓↓	↑↑↑	→↑	↑
Hyperventilation					
neurogenic	↑	→	↓↓↓	→	↑↑↑
DKA	↓↓	→	↓↓	↓↓↓	↑↑

For example, pulmonary edema in association with chronic obstructive airway disease will result in greater hypoxia with normal or elevated carbon dioxide tension due to associated hypoventilation. Similarly, if carbon dioxide tension is normal or elevated in association with pulmonary edema, the cause of hypoventilation, such as oversedation with morphine and diazepam or a central nervous system disorder,

Table 8.6: Indications for arterial blood gas estimation

Severe breathlessness
Pulmonary edema
Respiratory failure due to any cause
Severe pneumonia
Suspected pulmonary embolism
Severe bronchial asthma
Metabolic encephalopathy
Poisoning
Acidotic states
Diabetic ketoacidosis
Uremia
Metabolic acidosis of any cause
Alcohol intoxication
Electrolyte imbalance
Hypokalemia
Hyperkalemia
Shock of any cause

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needs to be investigated. Commonly, acute cardiac illness may precipitate diabetic ketoacidosis in a diabetic, thereby adding another factor in the interpretation of arterial blood gas analysis.

Apart from its role in diagnosis, arterial blood gas analysis is useful in making decisions about artificial ventilation and also in monitoring progress.

Though dyspnea is often identified with cardiac or pulmonary disease, a variety of medical disorders manifest or resemble dyspnea. Intelligent discrimination of one from the other greatly assists patient management and limits unnecessary laboratory testing.

PRACTICE IMPLICATIONS

- Aspiration pneumonia as a cause of dyspnea is often missed.
- In all patients with 'unexplained' dyspnea, arterial blood gases (ABGs) should be obtained. It is not rare for metabolic acidosis to be mistaken for dyspnea due to cardiac or pulmonary disease.
- Drowsiness or altered sensorium in a dyspneic patient should alert the clinician to the possibility of associated metabolic disorder (hypoglycemia, metabolic acidosis, drug induced) or cerebrovascular accident.
- Surprisingly, dyspnea as a symptom or as a physical sign may be missed.
- Pulmonary venous hypertension is not equivalent to dyspnea.
- Acute respiratory failure may be missed when weakness is the presenting symptom and dyspnea may not be obvious.

9 Syncope

Syncope is a sudden, transient loss of consciousness. It may be due to a trivial vasovagal episode or due to a life threatening arrhythmia. Unless proved otherwise syncope should be considered as an aborted sudden death. This dual significance makes it an important symptom to evaluate carefully. As the potentially serious causes are usually cardiac, the cardiologist or the physician is usually consulted.

While syncope is transient loss of consciousness, near syncope or pre-syncope is near loss of consciousness by lesser degrees of the same cause producing syncope. Patients use a variety of terms to describe this symptom, which can be misleading. The common descriptions are faint, blackout, spell, swoon and giddiness.

Syncope occurs either due to fall in cerebral blood flow or reduction in energy substrates to the brain. The mechanisms are:

Reduction in cerebral blood flow

Fall in central aortic pressure (< 60 mmHg, mean)

Elevation in cerebrovascular resistance or intracranial pressure

Reduction of energy substrates

Hypoxia ($PO_2 < 60$ mmHg)

Hypoglycemia

When the mean aortic pressure falls to below 60 mmHg in a normal person, cerebral perfusion falls significantly and syncope occurs. The blood sugar values are usually less than 40 mg% when syncope occurs due to hypoglycemia. The arterial PO_2 is usually less than 60 mmHg (saturation 80 per cent) before syncope occurs due to hypoxia. These values are applicable when a singular factor is operative. When more than one factor is operative, even lesser degree of abnormality can result in syncope (for example, hypoglycemia in a 70-year-old with cerebrovascular disease).

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Causes of syncope

Syncope could be due to vasovagal/psychogenic, cardiovascular, metabolic encephalopathy/drug related, and central nervous system disorders. Sometimes no attributable cause may be found. Table 9.1 shows the various categories and the relative frequency of each in the combined results from the three major prospective studies.

The numbers are a summation of the prospective studies on syncope. The largest single cause is surprisingly 'unknown cause'. This information is valuable in the diagnostic testing and advice to the patients with syncope.

VASOVAGAL/PSYCHOGENIC SYNCOPE

The vasovagal syncope is the most common form of syncope accounting for half of all the causes of syncope. It is also known as neurocardiogenic syncope. It could be triggered by:

- Acute severe pain due to any cause
- An unpleasant or distressing sight
- Prolonged standing
- Fasting state
- Crowded, suffocating, uncomfortable surroundings
- Micturition
- Defecation
- Swallowing
- Laughing
- Carotid sinus hypersensitivity
- During cardiac catheterization/pressure over groin

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Table 9.1: Incidence of major categories of syncope

<i>Diagnosis</i>	<i>No. of patients</i>	<i>Percentage</i>
Vasovagal/psychogenic	203	37
Cardiac	75	13
CNS	26	5
Metabolic/drugs	16	3
Unknown	230	43
Total	550	

- Postural hypotension
- Hyperventilation
- Psychogenic factors

Since a syncope may be associated with certain special situations like sudden severe pain, grief, humiliation or anger, death of a loved one, sight of blood, and so on, it led to the use of the term 'situational syncope'. It occurs more commonly in hot humid environments.

The typical vasovagal episode has two phases. In the first phase a steady fall in arterial pressure occurs with or without increase in heart rate. In the second phase, a sudden fall in heart rate occurs, resulting in an abrupt fall in arterial pressure. Atropine prevents the fall in heart rate but the vasodilatation during the first phase may be enough to cause hypotension and loss of consciousness. Blood flow to skeletal muscles increases but that of brain, kidney and mesenteric circulation decreases.

The typical vasovagal episode is described in Table 9.2.

Table 9.2: Features of a vasovagal syncope

Position	Usually standing or sitting but may occur in supine position (as during cardiac catheterization)
Prodrome	Few seconds to minutes Weakness, light-headedness, sweating, nausea, yawning, pallor or the sensation of impending doom Urge to pass urine or motion
The event	Loss of consciousness, with a fall to the ground or may just slump to one side while sitting Pallor, sweating, dilated pupils Pulse is slow or of low volume Urinary incontinence may occur Total duration is most often less than a minute, rarely 2–5 minutes
Post event	Awakens with a feeling of dizziness and nausea, sweating, and the urge to defecate Orients to surroundings within seconds after gaining consciousness Total duration of episode 5–10 minutes If tries to stand up immediately, may again fall

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The commonest form of vasovagal syncope is usually benign and is self limiting. The vasovagal episodes that occur during cardiac catheterization are generally related to the anxiety and fear, and pain related to groin puncture and pressure. These episodes are often promptly recognized as almost all these patients are closely monitored. They promptly respond to atropine and fluid load. Unrecognized, the hypotension of vasovagal episode may prove fatal in patients with severe obstructive coronary disease or in patients with limited cardiac reserve.

Rock concert syncope

Lempert and Bauer recently described this entity of mass fainting among rock concert audiences. The faints were classified as hyperventilation or panic attacks. In the majority, anxiety is provoked when they felt squeezed, choked, and trapped in the middle of the crowd whereas others start hyperventilating when they are overcome by emotion. A combination of syncope provoking factors were identified. These are sleeplessness during the previous night, fasting from early in the morning, when they first line up, long period of standing in the arena, and hyperventilation.

Rock concert syncope provoking factors causing cerebral vasoconstriction and Valsalva-like pressure induced either by screaming or reflexly by external compression of the thorax by the pushing masses. Valsalva maneuver impairs venous return to the heart and consequently reduces cardiac output. The authors gave a few guidelines to prevent this syncope, summarized as follows: sleep, eat, sit, keep cool, and stay out of the crowd.

SYNCOPE IN CARDIOVASCULAR DISORDERS

Though cardiovascular disease is often suspected as the first etiological possibility, these disorders are responsible for only 13 per cent of cases of syncope. In cardiovascular causes of syncope, arrhythmias account for nearly 80 per cent of cases. The various causes of syncope in cardiovascular disorders are listed in Table 9.3.

Arrhythmias produce syncope when the heart rate is too high or too low, to sustain cardiac output and arterial pressure. The commonest arrhythmia producing syncope is ventricular tachycardia.

Table 9.3: Causes of syncope in cardiovascular disorders

<i>Arrhythmias</i>	
Bradyarrhythmias	Sinus bradycardia Acute ischemic syndromes Sick sinus syndrome Hyperkalemia Acute intracranial hypertension
	Complete heart block
	Congenital
	Acquired
	Acute ischemic syndromes
	Drug induced
	Degenerative disease
	Hyperkalemia
	Diphtheritic myocarditis
Tachyarrhythmias	Supraventricular
	Ventricular
Conditions predisposing to arrhythmias	Myocardial ischemia Ventricular aneurysm Structural defects with chamber enlargements Mitral valve disease Atrial septal defect Ebstein's anomaly of tricuspid valve Heart failure Hypokalemia Hyperkalemia Prolonged QT syndromes Pre-excitation syndromes Renal failure Severe emotion Carotid sinus hypersensitivity Intramyocardial tumours
Structural defects	Severe aortic stenosis Hypertrophic cardiomyopathy Acute ischemic syndromes Acute pulmonary embolism Cardiac tamponade Primary pulmonary arterial hypertension Mitral stenosis Severe pulmonary stenosis Atrial myxoma Acute myocarditis Tetralogy of Fallot (severe) Eisenmenger syndrome

Ventricular tachycardia (VT)

This arrhythmia accounts for about 40 per cent of cardiovascular causes of syncope. In elderly patients beyond 70 years, this is the most common diagnosis and accounts for 25 per cent of all episodes of syncope. Almost all patients with ventricular tachycardia have underlying heart disease. Most of the patients have no prodromal symptoms. A brief episode of dizziness or light headedness may be experienced. A preceding history of palpitation may occur. As a rule, most of these patients lose consciousness within a few seconds and a history of several minutes of warning symptoms generally suggests a vasovagal syncope. The diagnosis of ventricular tachycardia is often established during the initial evaluation of the patient either by the 12-lead ECG or monitoring.

Long QT syndrome

Prolongation of QT interval occurs due to a variety of causes and predisposes to a peculiar form of ventricular tachycardia called *torsade de pointes*.

Idiopathic long QT syndrome is common. There are bizarre morphologies of the T waves. Syncope or cardiac arrest may occur under physical or emotional stress. These patients are often misdiagnosed as having epilepsy. The majority of patients have their first syncope before the age of 20 years. If left untreated, mortality is high due to *torsade de pointes* degenerating into ventricular fibrillation. Antiadrenergic interventions by either betablockers or left cardiac sympathetic denervation are very effective in ventricular fibrillation and sudden death. With treatment, the 5 year mortality is below 4 per cent. Untreated, mortality is 20 per cent within the first year after the syncope and is 50 per cent within 10 years. The diagnosis would not be missed, provided one interprets the ECG carefully in a case of syncope evaluation.

Supraventricular tachycardia (SVT)

Syncope is less common with this arrhythmia in comparison to ventricular tachycardia and accounts for 8 per cent of all the causes of syncope of cardiovascular origin. Presence of certain factors predisposes to syncope:

- Underlying heart disease/failure
- Aortic stenosis
- Hypertrophic cardiomyopathy
- Pulmonic stenosis

- Restrictive cardiomyopathy
- Rate of SVT ($> 200/\text{min}$)
- Underlying pre-excitation
- Age (advanced age)
- Standing position at the time of onset of SVT

The underlying heart disease has to be severe to produce syncope with supraventricular tachycardia. When the rates are exceedingly high, as in pre-excitation, diastolic filling is impaired and the cardiac output and the arterial pressure fall to below the critical level. Advanced age, with associated cerebrovascular disease, may predispose to syncope even with a slight fall in arterial pressure. Paradoxically, younger individuals with better AV nodal conduction and more ventricular rates may be more susceptible to syncope than the elderly with slow AV nodal conduction.

Bradyarrhythmia

Bradyarrhythmias and advanced AV blocks account for about 31 per cent of all the cardiovascular causes of syncope; complete heart block and sick sinus syndrome (SSS) account for the majority of cases. More than one mechanism may be responsible for syncope in sick sinus syndrome.

- Severe sinus bradycardia
- Complete heart block
- Sinus arrest either spontaneous or due to post SVT pause.
- SVT/AF with rapid ventricular rates
- SVT/AF degenerating into VT/VF
- Verapamil or betablocker therapy

Following the relief of a sudden onset palpitation if the patient has syncope, sick sinus syndrome should be suspected. It is important to rule out sick sinus syndrome in all patients presenting with supraventricular tachycardia because verapamil given for supraventricular tachycardia can aggravate bradyarrhythmia and may result in a fatal asystole. In any supraventricular tachycardia with heart rate less than 200 per minute but recurrent syncope and a normal heart, suspect sick sinus syndrome.

Complete heart block

Syncope is rare in patients with congenital complete heart block (CHB) as the

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ventricular rates are usually adequate. Most of the patients with syncope due to complete heart block have some form of conduction defect. The ECG features at the time of admission are:

- RBBB + Left anterior hemiblock
- Alternating bundle branch block
- RBBB with first degree AV block
- LBBB with first degree AV block
- Fascicular blocks with Mobitz II AV block

In syncope due to complete heart block, prodromal symptoms are often absent. Unconsciousness supervenes within 5 seconds in standing position or within 15 seconds in recumbent position. Bowel or bladder incontinence with fits may occur and a diagnosis of seizure disorder is often made initially. In a patient with syncope, if the ECG shows one of the changes mentioned above, complete heart block as a cause of syncope should be considered. In a study by Dhingra et al, 200 patients with chronic bifascicular block were studied; syncope occurred in 30 patients (15 per cent). In 5 of them complete heart block was documented. A pacemaker was needed in only 6 of the 30 patients (20 per cent). The majority of patients had syncope due to ventricular arrhythmia or unknown cause. This illustrates the need to use caution and judgment in evaluation of patients with syncope and a seemingly abnormal ECG.

To a quick question give a slow answer.

Italian proverb

RENAL FAILURE PRESENTING WITH BRADYCARDIA AND SYNCOPE

Renal failure is generally not considered a diagnostic possibility in patients presenting with syncope. The **mechanisms** of syncope in renal failure are:

- Hyperkalemia (bradycardia)
- Drug induced bradycardia (atenolol)
- Postural hypotension (drug induced)
- Hypertensive encephalopathy
- Pericardial tamponade
- Gastrointestinal bleeding
- Hypermagnesemia (magnesium containing antacids)

The first two categories deserve special mention as they are often missed in practice.

Case summary

A 50-year-old housewife was admitted for evaluation of syncope and was detected to have complete heart block. She was referred for a permanent pacemaker implantation by a cardiologist. Initial evaluation revealed her to be in complete heart block with a ventricular rate of 35 per minute and a narrow QRS complex. The blood pressure was 120/80 mmHg. A temporary pacemaker was inserted with a view to implant a permanent one selectively. Serum electrolytes including potassium were reported as normal. Another routine sample of serum electrolytes showed serum potassium of 7 meq. The creatinine was 9 mg%. She was hemodialysed with regression of complete heart block and improvement of renal parameters and was discharged home without a pacemaker.

This patient illustrates the need to obtain serum electrolyte levels as a routine in all patients presenting with arrhythmia and the need to recheck the values more than once if the clinical features warrant it. In this patient, the tall T-waves were a possible clue. Even if this was missed, the drowsy patient with well maintained blood pressure is a pointer to a metabolic encephalopathy like abnormal glycemic state or renal failure. Elevated creatinine value was a clue to the underlying hyperkalemia. If the laboratory error was not rechecked this patient would have had a fatal outcome.

Case summary

A 61-year-old retired police officer started having recurrent syncope at rest and on exertion. He was referred for bradycardia and heart failure. He was known to have hypertension and diabetes mellitus. His serum creatinine was found to be 2.4 mg% six months prior to admission. He was given atenolol 50 mg per day along with glybenclamide for diabetes. Evaluation at presentation revealed complete heart block with a ventricular rate of 40/min, mild left ventricular failure and serum creatinine of 3 mg%. He improved with restoration of sinus rhythm after 4 days of withholding atenolol.

This case illustrates the danger of prescribing atenolol in patients with renal failure. Even routine doses accumulate and cause life threatening bradycardia. This is such a common occurrence that the authors recommend routinely checking serum creatinine levels again whenever there is unexpected bradycardia with atenolol.

NON-ARRHYTHMIC CARDIOVASCULAR CAUSES OF SYNCOPE

AORTIC STENOSIS (AS)

Among valvular lesions, aortic stenosis is the commonest defect responsible for syncope. In three prospective studies of 550 patients with syncope only 6 (1.09%) had aortic stenosis. In aortic stenosis, syncope is generally suggestive of severe obstruction or arrhythmia. The **causes** could be

- Exertional syncope due to severe aortic stenosis
- Arrhythmias
- Supraventricular
- Ventricular
- Bradyarrhythmias
- Associated coronary artery disease
- Unrelated to AS

Exercise induced syncope is one of the three principal symptoms of aortic stenosis and occurs in two stages. The first stage is heralded by sudden fall in blood pressure, pallor, inattention and light headedness. This is followed by loss of consciousness. The ECG during this stage is normal. If the episode lasts more than a few seconds, the second stage may supervene. Most of the features of the second stage are secondary to severely reduced coronary blood flow.

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First stage (20–40 seconds)

Sudden fall in blood pressure
 Pallor
 Light headedness
 Inattention
 Unconsciousness
 Sinus rhythm
 Reduced intensity of heart sounds

Second stage

Cyanosis
 Absent pulse and heart sounds
 Apnea
 Twitching of body or seizure

Bladder or bowel incontinence
Abnormal ECG
Sinus tachycardia
Ventricular tachycardia
Ventricular fibrillation
Atrial fibrillation
AV block
Asystole

Mechanisms of exertional syncope in aortic stenosis

The normal cardiovascular response to exercise is characterized by fall in systemic vascular resistance with increase in cardiac output so that the central aortic pressure is maintained. When a patient with severe aortic stenosis exercises, the exertional fall in systemic vascular resistance is not counteracted by a rise in cardiac output. As a result, the blood pressure falls and syncope occurs. The reflexes (Bezold-Jarisch reflex) from the left ventricle under increasing pressure of exercise may also play a role. Normally, the leg exercise is accompanied by a reflex vasoconstriction in the forearm. When a patient with severe aortic stenosis exercises, the high intraventricular pressures reflexly inhibit forearm vasoconstriction and a precipitous fall in arterial pressure occurs.

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The mechanisms are:

- Fall of systemic vascular resistance (vasodilation in skeletal muscles)
- Failure of forearm vasoconstriction during leg exercise (ventricular Bezold-Jarisch reflex)
- Cardiac output fails to rise due to severe fixed obstruction

Once syncope occurs in aortic stenosis, most of the patients do not survive longer than 3 years. After the severity of aortic stenosis is assessed by echo-Doppler studies, cardiac catheterization should be done to confirm it and surgery should be offered as early as possible. If syncope is not accompanied by angina or dyspnea, or there is a discrepancy between the clinical examination, ECG, and echo-Doppler, cardiac catheterization should be performed to estimate the severity of stenosis. If aortic stenosis is found to be non-severe, some other cause for syncope is likely, such as coronary artery disease, hypertrophic cardiomyopathy, or arrhythmic, non-cardiac cause.

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Exercise thallium or coronary arteriogram help in detecting coronary artery disease. In hypertrophic cardiomyopathy, syncope occurs independent of the severity of outflow obstruction, and is related to restriction to ventricular filling or arrhythmias.

CORONARY ARTERY DISEASE (CAD)

Though traditional teaching focuses on aortic stenosis as an important cause of syncope, coronary artery disease probably is the commonest cause of syncope in middle aged and elderly patients. Multiple mechanisms are responsible for syncope in coronary artery disease. The causes are:

Arrhythmias

- Bradyarrhythmias

- Tachyarrhythmias

Large area of myocardium at risk

- Acute myocardial infarction

- Acute ischemia

Parasympathetic excess

- Acute inferior MI

Drug induced

- Vasodilators (nitrates, nifedipine, morphine)

- Volume depletion (diuretics)

- Arrhythmias (dopamine, isoproterenol, digoxin, antiarrhythmic agents, proarrhythmia).

Onset of acute mechanical complications

- Ventricular septal defect

- Mitral regurgitation

- Ventricular rupture

- Acute pulmonary embolism

- Cardiac tamponade

- Postinfarct pericarditis (thrombolysis, anticoagulants)

- Perforation by pacemaker wire

- During coronary angioplasty

Postural hypotension after prolonged bed rest

- Vasospastic angina (ventricular arrhythmia is common)*

- During and after coronary arteriography*

Syncope with chest pain suggestive of myocardial ischemia usually suggests a polymorphic ventricular tachycardia or ventricular fibrillation or large area of myocardium at risk resulting in hypotension and is an indication for aggressive investigation and management. In the early hours of acute myocardial infarction, syncope with a history of fall should make one look for evidence of injury to the head or blunt trauma to abdomen as thrombolysis may precipitate serious hemorrhage in these patients.

Case summary

A 34-year-old hypertensive engineer presented with chest pain of 2 hours duration and the ECG revealed an evolving inferior myocardial infarction with sinus rhythm. Prior to hospitalization, he had syncope once. There were no obvious signs of external injury to the head or any other site. Pulse was 70/min and BP 150/100 mmHg. The rest of the physical examination was unremarkable. Intravenous streptokinase 1.5 million units was given. Two hours after thrombolysis, he became drowsy and later became unconscious. Bogginess and hematoma appeared over the left frontoparietal region of the scalp. A CT scan revealed diffuse intracerebral hemorrhage and he died 24 hours later.

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This patient illustrates the need to be cautious in patients with a history of syncope before instituting thrombolytic therapy. On careful analysis, this patient probably should have had a thorough examination to rule out head injury and should not have received thrombolysis directly as the infarction was inferior with a small area of myocardium at risk and the patient was a hypertensive. The evidence of external injury to the head was obviously missed.

Nitrate syncope

Sublingual nitrate can produce syncope by sudden reduction of venous return due to venodilation and is often counteracted by reflex tachycardia (Table 9.4). Concomitant therapy with diuretics, vasodilators and betablockers increases the tendency for syncope. A new onset of nitrate syncope in a patient with chronic stable angina may mean progression of the disease to unstable angina or evolving myocardial infarction. Such a history should be used as a clue for prompt hospitalization and management.

The indication for temporary pacing should be more liberal when the patient with acute MI presents with syncope.

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Table 9.4: Factors predisposing to 'nitrate syncope'

<i>Factor</i>	<i>Mechanism</i>
Progression to unstable angina/ acute MI	Unrelieved ischemia Additional area of ischemia Bradycardia (inferior MI)
Diuretics Vasodilators	Hypovolemia with venodilation Additional vasodilatation
Betablockers	Prevents reflex tachycardia
Prolonged standing	Venous pooling

OTHER LESIONS CAUSING SYNCOPE

Apart from aortic stenosis, any severe obstruction to circulation may cause syncope. Syncope is not uncommon in severe primary pulmonary arterial hypertension. It can be the presenting feature of acute pulmonary embolism. Syncope is rare in mitral stenosis even when it is severe. Recurrent syncope in a patient with mitral stenosis should suggest a left atrial myxoma as a cause of mitral obstruction. Rarely, paroxysmal atrial arrhythmias, severe pulmonary arterial hypertension, acute pulmonary or cerebral embolism may cause syncope in mitral stenosis. Associated aortic stenosis may also be the cause of syncope.

Ball valve thrombus though very rare, is more easily detected now with echocardiography. The patient may present with syncope or shock and can be relieved of the obstruction by elevation of the foot end of the bed.

Syncope in mitral stenosis: The causes could be:

- Untreated critical disease
- Left atrial myxoma
- Paroxysmal atrial arrhythmias
- Severe pulmonary arterial hypertension
- Cerebral embolism
- Pulmonary embolism
- Ball valve thrombus
- Associated aortic stenosis
- Associated coronary artery disease
- Cardiac tamponade during balloon mitral valvuloplasty

Systemic hypertension and syncope: Syncope in a patient with hypertension should suggest postural hypotension due to drugs (methyl dopa), hypertensive encephalopathy, pheochromocytoma or cerebrovascular accident. The causes could be due to:

- Postural hypotension
- Drug induced (methyl dopa)
- Acute intermittent porphyria
- Hypertensive encephalopathy
- Pheochromocytoma
- Intracranial hemorrhage (cerebellar)

Syncope during and following cardiac catheterization: Hypotension can occur during or after the procedure. The commonest cause remains vasovagal. The typical patient is anxious, and is either not informed or is too well informed about the procedure. Syncope often occurs at the time of groin punctures. This should be suspected when the patient becomes restless, starts yawning and may vomit. The fall in blood pressure may not be noticed until later as the arterial sheath is not yet inserted. They respond promptly to atropine if given early. Though vasovagal episodes are self limiting and respond easily to atropine in the majority of patients, in rare cases it can be fatal. A fatal outcome is more likely in a patient with underlying severe cardiac disorder (left main disease, severe triple vessel disease, or severe aortic stenosis, primary pulmonary arterial hypertension or severe congestive heart failure). I know of a patient who had normal coronary arteries and normal intracardiac pressures who died later in the ward of a vasovagal episode which was not recognized early. Proper hydration of the patient would prevent many episodes of vasovagal syncope.

Once hypotension occurs in patients with left main disease, coronary perfusion falls leading to myocardial ischemia and further hypotension, which prevents any recovery. An episode of vasovagal attack may be disastrous in this setting. The pain associated with groin compression during sheath removal may result in vagal episodes. In post-PTCA patients, femoral sheaths should be removed under the surveillance of trained personnel. Prophylactic intravenous atropine prevents the vagal episodes. Adequate sedation and analgesia are important. The patients should be monitored for their rhythm and blood pressure during the removal and immediately afterward.

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Vasovagal episode of cardiac catheterization/PTCA

The setting

- Anxious, fearful patient
- During femoral punctures
- During sheath removal

Potentially serious in

- Left main coronary disease
- Severe triple vessel disease
- Severe AS
- Severe pulmonary arterial hypertension
- Post-PTCA (abrupt vessel closure)

Prophylactic measures

- Adequate explanation to the patient regarding the procedure.
- The atmosphere of the catheterization lab and the attitude of staff.
- Intravenous atropine in anxious bradycardiac patients particularly prior to sheath removal.
- Adequate hydration and appropriate sedation.

NEUROLOGICAL DISORDERS WITH SYNCOPE

Any episode of syncope is most commonly believed to be and is confused with epilepsy. This is more likely when the classic signs and symptoms are not obvious. Other neurological disorders that may mimic syncope are:

- Epilepsy
- Akinetic seizures in adults
- Petit mal seizures in adolescents/children
- Partial complex seizures
- Generalized tonic clonic seizures
- Narcolepsy/cataplexy
- Transient ischemic attacks (posterior territory)
- Loss of consciousness
- Drop attacks
- Subarachnoid hemorrhage
- Basilar artery migraine
- Bulbar syncope

Complex partial seizures are often mistaken for syncope. In one study, 25 per cent of this type of seizures arising from temporal and deep frontal structures

presented with staring spells and loss of consciousness without other signs of seizure disorder. Visceral symptoms like abdominal discomfort, nausea and vomiting occur in 40 per cent of patients. Symptoms of sympathetic excess such as anxiety, fear, sensation of impending doom, flushing, tachycardia and hypertension are common. An erroneous diagnosis of vasovagal syncope or cardiovascular disorder causing syncope is often made. Complex partial seizures may mimic angina, cardiac arrhythmia or pheochromocytoma. In a recent study almost half of the patients were shown to have an arrhythmia during the seizure. Drop attacks are probably caused by brainstem ischemia. Consciousness is retained during the episode. Basilar artery migraine is possibly caused by vasospasm of the posterior cerebral circulation. The accompanying typical migrainous symptoms help in the differentiation. Bulbar syncope is caused by any brainstem lesion (tumours, previous infarcts, amyotrophic lateral sclerosis, syringobulbia) that regulate heart rate and vasomotor functions. Unconsciousness of psychogenic origin is common and usually occurs in the presence of an audience. The person never hurts himself when falling and the accompanying pallor, flushing and other autonomic disturbances are absent.

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It is difficult to differentiate a syncope from a seizure (Table 9.5). An abrupt loss of consciousness, aura, and prolonged period of amnesia favour a diagnosis of seizure.

Table 9.5: Differentiation of syncope from seizure

<i>Features favouring seizure</i>	<i>Features common to both</i>
Aura	Blackout or loss of vision
Abrupt loss of consciousness	Giddiness
Sensory hallucinations	Nausea
Deja vu experiences	Sweating
Emotional states	Weakness
Confusional states	Yawning
Motor automatism	Sighing
Prolonged amnesia	Pallor
History of alcohol withdrawal, head trauma, cerebrovascular accident	
Recurrent syncope in young healthy person	
Known malignancy (potential for secondaries in brain)	

Carotid disease: Unilateral carotid disease is accompanied by symptoms of hemispheric ischemia. Bilateral carotid stenosis (as in Takayasu's arteritis) can result in exertional syncope like aortic stenosis. In elderly patients who frequently have an ejection systolic murmur due to aortic valve sclerosis, this can be mistaken for aortic stenosis. However, the murmur being better audible in the carotids than over the precordium or aortic area is a helpful indicator.

Vertebrobasilar insufficiency (VBI): Syncope due to this entity is more common in patients of age 65 years and older. Associated diplopia, ataxia, dysarthria and sensorimotor disturbances are common. Though atherosclerosis is the commonest underlying cause, cervical spondylosis, subclavian steal syndrome, and congenital anomalies of cervical vertebrae may be responsible. The bony abnormality can obstruct blood flow.

Subarachnoid hemorrhage (SAH): Sudden loss of consciousness or syncope can occur in subarachnoid hemorrhage due to sudden rise of intracranial pressure with recovery of sensorium either fully or partially. Preceding severe headache though common, is not often remembered by the patient, due to retrograde amnesia. Hypertension and bradycardia are common. The ECG may show ST-T alterations which can mimic unstable angina, subendocardial infarction or anterior myocardial infarction. Arrhythmias can also occur. Careful evaluation often reveals slight alteration in sensorium, neck rigidity or other neurological signs. A CT scan should be asked for in case of doubt.

Case summary

A 60-year-old hypertensive man, was admitted for evaluation of syncope. He was fully conscious at admission to ICCU. Pulse: 66/minute; BP:170/110 mmHg; S4 was audible. ECG showed LVH with strain and deep symmetrical T wave inversions with ST depressions all over the chest leads. A diagnosis of acute 'silent' subendocardial infarction was made. He was given aspirin and intravenous heparin. After 24 hours he was frankly unconscious and developed a full blown clinical picture of subarachnoid hemorrhage confirmed by CT scan. He died a month later.

This patient illustrates how subarachnoid hemorrhage can mimic acute coronary syndromes. This entity deserves special mention for more than one reason. Firstly, it is not generally realized that subarachnoid hemorrhage is capable of transient loss of consciousness with recovery, only to recur later. Secondly, patients

with undiagnosed syncope often go to an intensive coronary care unit or a cardiologist. Thirdly, subarachnoid hemorrhage mimics acute ischemic syndromes by the ECG changes that it can produce. Finally, antiplatelet agents, anticoagulants or thrombolytic therapy given in this setting can be disastrous.

Common reasons for mistakes in diagnosis or treatment are:

- The presumption that syncope is almost always cardiovascular in origin
- Lack of knowledge that syncope occurs in SAH
- Most patients with syncope go to coronary care units or cardiologists
- The ECG changes (ST depressions, deep T wave inversion) occurring in SAH mimic acute ischemic syndromes
- The esophageal pain that follows vomiting mimics the chest pain of myocardial ischemia
- Present tendency for a lower threshold to prescribe antiplatelets, anticoagulants, and thrombolytic agents

Case summary

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A 55-year-old businessman presented with chest pain and vomiting 3 hours prior to admission. The blood pressure was 170/100 mmHg and the pulse 70/minute. The rest of the physical examination was 'unremarkable'. The ECG showed features suggestive of acute subendocardial infarction and he was prescribed aspirin, heparin nitroglycerine and diltiazem. He complained of severe headache while in the CCU (this was attributed to nitrates). Four hours after admission, he became drowsy and a diagnosis of subarachnoid hemorrhage was confirmed on CT scan. Luckily this patient survived all this and was discharged without neurological deficit. On careful questioning, the patient admitted to having had a headache first followed by vomiting. The chest pain followed three vomitings.

Syncope in intracerebral hemorrhage: In intracerebral hemorrhage, syncope can occur but some alteration in sensorium is a rule and the diagnosis is easier. Cerebellar hemorrhage often presents as syncope and the associated severe hypertension and ECG changes may be misleading.

Brain tumours presenting with syncope: Tumours of the third ventricle (colloid cysts), those involving the foramen of Monro, and both frontal lobes can present with recurrent syncope without any definitive evidence of seizure activity. Posterior fossa tumours or malformations may present with loss of consciousness during episodes of coughing or sneezing related to cerebellar tonsillar herniation. This

entity may be dismissed as cough syncope and the dangerous underlying condition may be missed. Rarely, brainstem tumours can present as postural hypotension.

Peripheral neuropathies presenting with syncope: Many peripheral neuropathies with associated autonomic neuropathy can cause postural hypotension and syncope. Other causes are:

- Diabetic neuropathy
- Alcoholism
- Guillain-Barre syndrome
- Amyloidosis
- Familial dysautonomia
- Drug induced
- Vincristine
- Cisplatin
- Acute neuralgias with pain eliciting vagal reflex
- Glossopharyngeal neuralgia
- Trigeminal neuralgia

Shy-Drager syndrome (primary autonomic insufficiency and parkinsonism) can also present with syncope.

Metabolic and drug induced syncope: The most common of these is hypoglycemia induced by insulin or oral drugs; rarely insulinoma may be responsible. Alcoholics lose consciousness due to central nervous system effects of alcohol but the autonomic neuropathy may also be responsible.

In actual practice, postural hypotension is most likely when a vasodilator is combined with a diuretic in the initial prescription. Other causes are:

- Alcohol intoxication
- Porphyria
- Drug induced postural hypotension
 - Methyl dopa
 - Diuretics
 - Vasodilators
 - Nitrates
 - Prazosin hydrochloride
 - ACE inhibitors
 - Calcium blockers
 - Nifedipine

Gastrointestinal and abdominal causes of syncope: Internal hemorrhage is the most important cause in this group of disorders. Other intra-abdominal causes are:

- Upper GI bleeding
- Exaggerated vagal reflex
- Vomiting
- Micturition syncope
- Endoscopy
- Barium enema (visceral distension)
- Any acute severe abdominal pain
- Acute perforation of a viscus
- Blunt trauma to abdomen with
 - Rupture of spleen
 - Rupture of liver
- Acute pancreatitis
- Liver abscess bursting into pericardium/pleura

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Upper gastrointestinal bleed presenting as syncope is often missed. The obvious evidence of bleeding by hematemesis and melena may be altogether absent. As much as 500 ml of blood can be sequestered in the gastrointestinal tract without either of these signs apparent. Abdominal pain may be absent, or it may be difficult to distinguish upper abdominal pain from lower chest pain. The use of aspirin, heparin and thrombolytic agents can be dangerous.

Case summary

A 60-year-old businessman had palpitation, syncope and chest discomfort. He went to the emergency room 30 minutes after the episode. There was no pallor. The blood pressure was 150/100 mmHg and pulse was 120/min. There was no abdominal tenderness. The ECG showed sinus tachycardia with ST segment depression and T wave inversion in inferior and lateral chest leads. A diagnosis of unstable angina was made and he was given aspirin, heparin, nifedepine and betablockers. Four hours after admission, the BP fell to 90 mmHg, with a pulse rate of 120/min. The BP came up to 120/80 mmHg with fluid load, but started falling again. At this stage, he vomited 200 ml of altered blood. Ryle's tube aspiration revealed 500 ml of fresh blood. Blood transfusion, antacids and ranitidine were given. Endoscopy confirmed an actively bleeding peptic ulcer. He was managed conservatively and was discharged home 2 weeks later. Six weeks after discharge, the exercise test was found to be normal.

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Table 9.6: Syncope of GI bleeding: misleading features

<i>Feature</i>	<i>Mechanism</i>
Syncope	Transient hypotension due to sudden loss of blood counteracted by sinus tachycardia, peripheral vasoconstriction.
Postsyncope hypertension	Reactive hypertension due to adrenergic excess
Lack of hematemesis/malena	500 ml of blood can be sequestered in GI tract without external evidence of bleeding
Lack of abdominal pain	Bleeding into gastrointestinal tract is usually painless
Chest discomfort	Esophagitis
ECG signs of ST depression, T wave inversion	Hypotension/tachycardia may trigger myocardial ischemia with associated CAD in older patient Associated left ventricular hypertrophy of hypertension

Acute gastrointestinal bleeding presenting with syncope is often mistaken for a cardiovascular disorder. That the hypotension can be transient (due to gastrointestinal bleeding) is often not realized (Table 9.6).

When the clinical picture is not typical in acute ischemic syndromes, one should defer the use of aspirin and heparin, until a Ryles tube aspiration is negative for gastrointestinal bleed. Rectal examination may help to detect the condition.

Unknown cause: After initial evaluation, as many as 42 per cent of patients may fall under this category. Though recurrent syncope is rare in this group, one year mortality is as much as 6 per cent in them. In elderly patients, the prognosis must still be guarded as many of them tend to have transient ischemic attacks or cerebrovascular accidents later. Incidence of sudden death may be 5 per cent or higher in the elderly age group. In all elderly patients with syncope of unknown origin, a possible cardiac or neurological cause should be sought by careful evaluation and follow up.

APPROACH TO A PATIENT WITH SYNCOPE

History and physical examination help to diagnose 75 per cent of patients with syncope.

Table 9.7: Important aspects in the history of a patient with syncope

<i>Feature</i>	<i>Significance</i>
Clinical background	
Diabetic patient	Hypoglycemia Coronary artery disease Cerebrovascular accident Transient ischemic attack Postural hypotension due to autonomic neuropathy
Hypertension	Postural hypotension (drugs) Encephalopathy Intracranial hemorrhage Transient ischemic attack Coronary artery disease
Alcoholism	Intoxication Autonomic neuropathy
CAD	Arrhythmias Acute ischemic syndromes Mechanical complication Drugs
Postoperative or prolonged rest	Pulmonary embolism Postural hypotension
Peptic ulcer	Gastrointestinal bleeding Acute perforation
The deaf patient	Prolonged QT syndrome
Painful/unpleasant event/sight	Vasovagal
History of fall or head injury	Subdural hematoma
Related to a physiological act	
Exertional syncope	Left ventricular outflow obstruction; Bilateral carotid stenosis (Takayasu's arteritis)
Syncope with upper limb exercise	Subclavian steal syndrome
Syncope with cough	Cough syncope
Syncope with micturition	Micturition syncope
Syncope with neck turning	Carotid sinus hypersensitivity

Careful physical examination gives valuable clues in some patients with syncope.

In general, if the patient with syncope is young, and the features are suggestive of vasovagal syncope, tilt test may be done, he/she should be reassured and

SYNCOPE

Table 9.8: Important aspects of physical examination

<i>Feature</i>	<i>Could indicate</i>
Drowsiness/altered sensorium	Hypoglycemia Central nervous disorder
Pulse rate	Vasovagal Arrhythmia
Blood pressure	Postural hypotension Hypertension Reactive hypertension following a fall
Tongue (bruise, bleeding)	Epilepsy
Head injury	Subdural hematoma
Carotid bruit	Transient ischemic attack
Heart	
S4, murmurs	Aortic stenosis, or other valvular lesions
Non-ejection click	Mitral valve prolapse
Irregular rhythm	Arrhythmia
Pericardial rub	Cardiac tamponade
Leg veins/calf muscles	Deep vein thrombosis with pulmonary embolism
Rectal examination/stool heme test	Occult gastrointestinal bleeding
Neurological examination	Neurological cause
Provocative maneuvers	Tests to bring out vagal instability
Carotid sinus massage	
Hyperventilation	
Valsalva maneuver	
Ocular compression	

Table 9.9: Diagnostic testing in syncope

<i>Test</i>	<i>Mechanism</i>
Blood sugar	Hypoglycemia
Hemoglobin, PCV	Low level indicates bleeding
Serum potassium	Arrhythmias due to high or low levels
Serum bicarbonate	Low level suggests seizure occurred
Blood gases	Hypoxia indicates pulmonary embolism
Cardiac enzymes	Myocardial infarction
Electrocardiogram (2–8 % yield)	Arrhythmia Myocardial infarction

CLINICAL METHODS IN CARDIOLOGY

<i>Test</i>	<i>Mechanism</i>
Holter monitoring (2% yield)	Unstable angina Vasospastic angina Hyperkalemia Hypokalemia Pre-excitation Prolonged QT interval Cardiac tamponade Acute pulmonary embolism
Echocardiogram (1.3% yield)	Arrhythmia Valvular disease Pulmonary hypertension Intracardiac tumours Hypertrophic cardiomyopathy Cardiac tamponade Ventricular aneurysm LV dysfunction Wall motion abnormalities
Tilt test	Indicated to confirm a diagnosis of vasovagal syncope Usually no underlying heart disease
Electrophysiological study	Low yield if done in all patients VT, VF, Syncopal SVT or AF may be induced Useful in assessing sinus node function and Conduction system
Neurologic testing (EEG, CT, MRI)	Low yield

extensive diagnostic testing is not indicated. On the other hand, the syncope of ‘unknown cause’ in an elderly patient requires thorough evaluation and if the cause is not clear at the end, careful follow up is indicated. These patients have a 6 per cent one year mortality either due to cardiovascular or cerebrovascular cause. The patient and the family should be educated about the warning signals of heart disease and stroke. Invasive electrophysiologic testing may be indicated in this subset of patients.

10 Palpitation

If we had keen vision and feeling of all ordinary human life, it would be like hearing the grass grow, or the squirrel's heart beat, and we should die of that roar which lies on the other side of silence.

George Eliot

Palpitation is the uncomfortable awareness of the heart beat, and is usually related to an alteration in heart rate, rhythm or augmentation of contraction. Though it is classically suggestive of an arrhythmia, it occurs in a wide variety of disorders of cardiac and non-cardiac origin. Due to the commonness, and the dual significance of a life threatening arrhythmia on the one hand, and a trivial anxiety on the other, it requires a careful evaluation. Palpitation occurs due to a forceful cardiac contraction, or an abnormality of heart rhythm. Forceful heart contraction occurs in volume overloads due to excessive preload or all conditions with adrenergic excess. An arrhythmia produces palpitation either when the rate is too high, too low or is irregular.

Evaluation

A systematic approach to this symptom is useful in patient evaluation.

- Is it palpitation or some other symptom simulating it?
- Did it precede or follow the knowledge of heart disease?
- Is it persistent or paroxysmal?
- What is the nature of palpitation?
- What are the associated symptoms?
- Is there an extra cardiac cause for palpitation?
- Is the patient taking any drugs that produce arrhythmias?

The chest discomfort of myocardial ischemia and dyspnea can be confused

for palpitation. Some patients use these three terms to describe whatever happens from the upper abdomen to the lower jaw. When palpitation follows the knowledge of heart disease, it is more likely to be psychogenic. Multiplicity of symptoms and the setting under which it occurs make situational anxiety more likely.

Some serious disorders which can present as palpitation are:

- Arrhythmias
- Coronary artery disease: Angina, acute myocardial infarction
- Valvular heart disease
- High output states
- Internal hemorrhage (sinus tachycardia)
- Acute pancreatitis (sinus tachycardia)
- Acute pulmonary embolism (sinus tachycardia/dyspnea)

Paroxysmal palpitation is suggestive of an arrhythmia. Persistent palpitation is suggestive of a volume load like aortic regurgitation or a persistent arrhythmia like atrial fibrillation. Even in these situations, palpitation may not be experienced at rest but may manifest on exertion. If the palpitation is paroxysmal, and an arrhythmia is likely further enquiries are necessary.

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The clinical setting in which palpitation occurs gives clues to the nature and seriousness of the arrhythmia (Table 10.1). Knowledge of preexisting heart disease like mitral valve disease or Ebstein's anomaly of tricuspid valve suggests supraventricular arrhythmias. A pre-existing WPW syndrome suggests a re-entrant supraventricular arrhythmia. Digoxin is generally contraindicated in this situation, particularly with atrial fibrillation.

Causes of palpitations

Cardiovascular

- Regurgitant lesions (aortic, mitral and tricuspid regurgitation)
- Left to right shunts
- Hyperkinetic heart syndrome
- Prosthetic heart valves
- Electronic pacemaker
- Arteriovenous fistula
- Aortic aneurysm

Arrhythmias

- Rapid regular (paroxysmal atrial tachycardia)

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Rapid irregular (atrial fibrillation)
 Bradyarrhythmias (complete heart block)
 Normal rate but irregular (ventricular ectopics)

Non-cardiac

Hyperkinetic circulatory states
 Anemia
 Fever
 Thyrotoxicosis
 Anxiety
 Hypoglycemia
 Pheochromocytoma

Drugs

Sympathomimetic drugs
 Vasodilators
 Digitalis
 Tricyclic antidepressants

Diaphragmatic flutter

Migraine

Table 10.1: Clues to arrhythmia in the clinical setting

<i>Setting</i>	<i>Clues</i>
Coronary artery disease	Ventricular arrhythmias Conduction disturbances
Heart failure	Digitoxicity Hypokalemia of diuretics
Mitral valve prolapse	Ventricular or atrial arrhythmias
Deafness	Prolonged Q-T Ventricular arrhythmias
Antiarrhythmic drugs	Proarrhythmia Prolonged Q-T
Diuretics	Hypokalemia Ventricular dysfunction
A patient with depression on tricyclic antidepressants	Prolonged Q-T Ventricular arrhythmias
Thyrotoxicosis	Atrial fibrillation with rapid ventricular response

<i>Setting</i>	<i>Clues</i>
Pre-excitation	Re-entrant SVT AF with wide QRS/rapid rates
Mitral valve disease	Atrial fibrillation
Diabetic patient	Hypoglycemia
Bronchial asthma	Drug induced sinus or supraventricular tachycardia
Systemic hypertension	Vasodilators Pheochromocytoma

Nature of palpitation

The nature of palpitation may give clues as to the nature of the arrhythmia (Table 10.2). The associated symptoms are related to the disorder responsible for palpitation or to the arrhythmia (Table 10.3). Syncope is the most important symptom to enquire after as it may indicate a serious underlying arrhythmia or a large area of myocardium at risk since palpitation and chest discomfort may be confused with each other. Syncope in association with palpitation should be considered a danger signal. If recent, the patient should be hospitalized and

Table 10.2: Diagnostic clues to nature of palpitation

<i>Nature of palpitation</i>	<i>Clue</i>
Missing of a beat	Premature ventricular contraction
Thump in the chest	
Fullness in the neck	
Rapid regular palpitation	Sinus tachycardia Supraventricular tachycardia Ventricular tachycardia
Rapid irregular palpitation	Atrial fibrillation Atrial flutter with varying block Atrial tachycardia with varying block
Sudden palpitation with sudden cessation or asystole followed by syncope	The tachycardia-bradycardia of sick sinus syndrome
Rapid pulsations in the neck	Supraventricular tachycardia Ventricular tachycardia
Giddiness/syncope	Bradyarrhythmia Ventricular tachycardia

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evaluated. If it happened some time ago, the patient can be investigated on an out patient basis.

Dyspnea with palpitation may mean that an arrhythmia is responsible for heart failure, if palpitation preceded dyspnea. If palpitation followed dyspnea, it may mean heart failure precipitating arrhythmia or acute episode of asthma with atrial arrhythmias due to respiratory insufficiency, or acute pulmonary embolism with atrial arrhythmias. When dyspnea accompanies palpitation, intrinsic heart disease is likely. As dyspnea can be an anginal equivalent, underlying coronary artery disease remains a possibility.

Palpitation with dyspnea in patients with coronary artery disease usually indicates ventricular arrhythmia, and poor ventricular function and sudden death is common. Such patients require hospitalization and monitoring before antiarrhythmic drugs are administered.

Table 10.3: Associated symptoms in palpitation

<i>Symptom</i>	<i>Significance</i>
Syncope	Low cardiac output during arrhythmia Hypoglycemia Pheochromocytoma
Dyspnea	Heart failure due to arrhythmia Myocardial ischemia/infarction with LVF Acute pulmonary embolism Acute episode of severe asthma with respiratory insufficiency
Chest pain	Arrhythmogenic myocardial ischemia (chest discomfort follows palpitation) Angina or infarction with arrhythmia (chest discomfort precedes palpitation)
Polyuria following palpitation	Paroxysmal atrial tachycardia Paroxysmal atrial fibrillation
Sweating	Anxiety Arrhythmia with hypotension Angina/myocardial infarction Hypoglycemia
Diarrhea	Thyrotoxicosis Irritable bowel syndrome Hypokalemia induced arrhythmia

Chest pain following palpitation is due to arrhythmia precipitating angina. If chest pain precedes palpitation, myocardial ischemia precipitating arrhythmia is likely. Both brady- and tachyarrhythmias can cause angina: *bradyarrhythmias* by the mechanism of hypotension and increase in ventricular diastolic pressure which reduces the coronary perfusion pressure and *tachyarrhythmias* by increasing the myocardial oxygen demands and the often associated hypotension.

The **mechanisms** of angina in arrhythmias are

Tachyarrhythmias

Increased heart rate increasing myocardial oxygen consumption

Less diastolic time

Hypotension (coronary perfusion)

Elevated ventricular diastolic pressure (coronary perfusion pressure)

Bradyarrhythmias

Hypotension (coronary perfusion)

Elevated ventricular diastolic pressure

It is often not realized that a bradyarrhythmia can present as angina and can also be the presenting manifestation occasionally in sick sinus syndrome.

Paroxysmal atrial tachycardia or atrial fibrillation are often associated with polyuria during and immediately after the episode of tachycardia. This is possibly mediated by atrial natriuretic hormone released from the atria.

PALPITATION IN VARIOUS DISORDERS

PALPITATION IN NORMAL PEOPLE

Palpitation occurs in otherwise normal people with exercise, emotional excitability and the daily anxieties.

Many lamentable effects of this fear causeth in men, as to be red, pale, tremble, sweat; it makes sudden cold and heat to come all over the body, palpitation of the heart, syncope, etc. It amazeth many men that are to speak or show themselves in public assemblies, or before some great personages.

Robert Burton in *The Anatomy of Melancholy*

Palpitation is a very common feature of anxiety states. The multiplicity of symptoms and the precipitating factors help in identifying it. In acute anxiety states, palpitation may be the dominant but transient symptom and is usually relieved

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spontaneously or with reassurance. The chronic form of anxiety is relatively resistant to therapy and is called by various names such as

- Da Costa syndrome
- Soldier's heart
- Effort syndrome
- Neurocirculatory asthenia
- Functional cardiovascular disease
- Irritable heart

The signs and symptoms are often indistinguishable from other hyperkinetic circulatory states.

Symptoms and signs of palpitations in anxiety states

Symptoms

Palpitation
Shortness of breath (deep sighing respiration)
Chest pain or tightness
Weakness
Insomnia
Giddiness

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Signs

Sinus tachycardia
High pulse pressure
Excessive sweating
Hyperventilation
Hyperkinetic apical impulse
Hyperkinetic parasternal impulse
Ejection murmurs across right or left ventricular outflow tract
Non-specific ECG abnormalities (ST-T alterations, voltage LVH in thin people)

The chronic nature of the disorder, deep sighing type of respiration, and atypical type of pain of coronary artery disease distinguish this disorder from the diseases it so commonly simulates. This diagnosis should be made only after carefully excluding a variety disorders. The conditions mistakenly labelled as anxiety disorders are:

- Anemia
- Thyrotoxicosis

- Pheochromocytoma
- Mitral valve prolapse
- Coronary artery disease
- Acute pulmonary embolism
- Hyperkinetic heart syndrome
- Rabies
- Episodic asthma
- Hypoglycemia in diabetic patients
- Insulinoma with recurrent hypoglycemia
- Pancreatic carcinoma
- Hepatic cirrhosis
- Drug addiction with periods of withdrawal
- Chronic low-grade fever as in pulmonary tuberculosis
- Drug use
 - Smoking
 - Coffee
 - Sympathomimetic drugs
 - Alcohol
 - Thyroid medication

The conditions listed above should be carefully ruled out before labeling somebody as having palpitation.

Choose your specialist and you choose your disease

Anonymous

PALPITATION IN VALVULAR HEART DISEASE

Palpitation is often the initial and most dominant symptom in mitral and aortic regurgitation. Even if palpitation is significant, surgical intervention is almost never indicated due to this symptom. Dyspnea is often the basis for decision making. Palpitation is rare in aortic stenosis. In mitral stenosis, palpitation occurs late in the disease with the onset of atrial fibrillation. In the initial stages of the disease, the arrhythmia tends to be paroxysmal and later becomes persistent. Once established, palpitation may be experienced only on exertion. Recurrent supraventricular tachycardia or atrial fibrillation may precipitate pulmonary edema in patients with mitral stenosis.

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- Typically palpitation precedes dyspnea in such situations.
- Look for mitral stenosis in all patients with ‘unexplained’ atrial fibrillation
 - Indicates chronic severe mitral stenosis in young patients
 - Increased incidence of peripheral embolism
 - Indication for long term oral anticoagulation
 - Indication for transesophageal echocardiography to rule out left atrial thrombus
 - Indication for relief of mitral stenosis
 - Atrial fibrillation in elderly patients may occur even with mild mitral stenosis
 - Paroxysmal palpitation explains ‘unexplained’ peripheral embolism with sinus rhythm or recurrent pulmonary edema

If atrial fibrillation occurs in a young patient with mild mitral stenosis, some other associated cause should be looked for.

The causes of atrial fibrillation in a young patient with mild mitral stenosis are:

- Associated atrial septal defect
- Thyrotoxicosis
- Pre-excitation
- Recurrent pulmonary embolism
- Sick sinus syndrome

Inappropriate sinus tachycardia and palpitation is common in many patients with mild to moderate mitral stenosis with exercise. They respond well to betablockers, and intervention can be postponed for months to years.

PALPITATION IN CONGENITAL HEART DISEASE

The equivalent of palpitation in infancy and childhood is increased precordial motion noted by the parent. This, as a sign or symptom, may be the presenting feature in ventricular septal defect and patent ductus arteriosus in early childhood, but occurs later in life in atrial septal defect. Palpitation is uncommon in Tetralogy of Fallot or conditions with similar physiology. Palpitation as a dominant symptom in cyanotic heart disease should suggest Ebstein’s anomaly of tricuspid valve as they are predisposed to recurrent atrial arrhythmias due to large right atrium. In

cyanotic heart disease with increased pulmonary blood flow, palpitations may occur. Atrial arrhythmias are common after Mustard operation of redirecting the venous blood in transposition of great arteries.

PALPITATION IN SYSTEMIC HYPERTENSION

Recurrent palpitation and sweating in a patient with paroxysmal hypertension should suggest the possibility of pheochromocytoma. More commonly, palpitation in patients with hypertension is related to drug therapy with vasodilators or the mere knowledge of hypertension.

Causes

- Pheochromocytoma
- Isolated use of vasodilators
- Associated coronary artery disease
 - Palpitation as anginal equivalent
 - Arrhythmias
- Hypokalemia causing arrhythmia
 - Diuretic induced
 - Primary aldosteronism
- Psychogenic: after the knowledge of hypertension

Palpitation may be an anginal equivalent, and careful evaluation in high risk patients by detailed history and objective testing will be helpful.

All palpitations are not arrhythmias and many arrhythmias do not palpitate.

11 Fever in a Patient with Heart Disease

Fever is a characteristic sign of infection. The majority of patients who present with fever have viral or bacterial infections. Fever in a patient with cardiovascular disease has different connotations and should be treated with concern and respect. Fever in this setting may indicate serious infection of the heart as infective endocarditis or pericarditis. It may also be responsible for aggravation of existing disorder due to increased demands on the heart. Some of the terms commonly used in this context are defined in Table 11.1.

Table 11.1: Definitions of terms commonly used in relation to fever

Normal body temperature	Oral: Usually 37 °C (98.6 °F) Rectal: 0.6 °C higher The normal temperature varies with time of the day, and between individuals (range 36.4 – 37.2 °C, or 97.5 – 98.9 °F)
Diurnal variation	Lowest in the early morning Highest in the late afternoon or early evening (4.00–8.00 PM) Variation may be as much as 0.5 °C (1.0 °F) Diurnal variation is consistent for each person Lack of diurnal variation may suggest factitious fever or hypothalamic disorder
Fever	Oral temperature > 37.2 °C (99 °F) in the morning and > 37.7 °C (100 °F) in the evening
Chills and rigors	At the onset of fever an abrupt increase in core temperature occurs by means of vigorous muscle contractions called rigors, associated with cutaneous vasoconstriction and piloerection, called chills.
Lethal temperature	Less than 26 °C (78.8 °F) or more than 43 °C (109.4 °F),
Upper lethal limit	Temperatures above 41 °C (105.8 °F)

DISORDERS ASSOCIATED WITH FEVER

That infections declare themselves with fever is a common knowledge, but a variety of non-infectious disorders may be accompanied by fever.

The clinician taking care of patients with cardiovascular disease should be aware of all the mechanisms responsible for this important and commonly occurring symptom, so that it may be managed appropriately (Table 11.2).

PATTERNS OF FEVER

INTERMITTENT (HECTIC OR SEPTIC) FEVERS

These are characterized by wide fluctuations in temperature with the temperature returning to normal at least once during any 24 hour period.

Causes

- Malaria
- Pyogenic abscess
- Bacteremia
- Irregular use of antipyretics
- Miliary tuberculosis

DOUBLE FEVER SPIKE IN A SINGLE DAY

Causes

- Gonococcal endocarditis
- Miliary tuberculosis
- Kala azar

VARIANTS OF INTERMITTENT FEVER

a. Alternate day fever: Causes could be *Plasmodium vivax*, steroid withdrawal fevers (alternate day dosage schedules)

b. Fever spike every third day: Cause could be *Plasmodium malaria* infection.

FEVER IN A PATIENT WITH HEART DISEASE

Table 11.2: Some causes of fever

Infections	<ul style="list-style-type: none"> Bacterial Viral Rickettsial Chlamydial Parasites particularly protozoal Fungal
Vascular	<ul style="list-style-type: none"> Inflammation Phlebitis, arteritis Thrombosis Infarction (myocardial, pulmonary, cerebral)
Immune mechanisms	<ul style="list-style-type: none"> Connective tissue disorders Drug reactions All immunological reactions Acquired immunodeficiency syndrome (AIDS)
Neoplastic disorders	<ul style="list-style-type: none"> Hematopoietic Lymphoreticular Hypernephroma Carcinoma of pancreas Cancer lung Hepatoma Bone tumours Complications of malignancy <ul style="list-style-type: none"> Disseminated metastasis Obstruction, stasis, infection
Granulomatous disorders	<ul style="list-style-type: none"> Sarcoidosis Granulomatous hepatitis
Inflammatory bowel disease	<ul style="list-style-type: none"> Ulcerative colitis Crohn's disease Others (acute pancreatitis, hepatitis)
Mechanical trauma	<ul style="list-style-type: none"> Trauma Surgery
Hemolysis, myolysis	<ul style="list-style-type: none"> Hemolytic anemia Rhabdomyolysis
Acute metabolic disorders	<ul style="list-style-type: none"> Gout Porphyria Thyrotoxicosis Pheochromocytoma Fabry's disease

SUSTAINED FEVER (CONTINUOUS FEVER)

There is moderately sustained elevation of temperature with minimal fluctuations.

Causes

- Typhoid fever
- Pneumococcal pneumonia
- Brucellosis
- Tularemia
- Psittacosis
- Rickettsial infections

REMITTENT FEVER

The fluctuations in temperature are less dramatic than in intermittent fever and the temperature does not return to normal

Causes

- Acute viral respiratory infections
- Plasmodium falciparum malaria
- Mycoplasma pneumonia

RELAPSING (RECURRENT) FEVER

Periods of fever and normal temperature alternate cyclically. During febrile episodes, the fever may follow any pattern.

Causes

- Lymphomas
- Rat bite fever
- Berylliosis
- Dengue fever

The catabolic response may impose serious hemodynamic burden on the cardiovascular system. A normal cardiovascular system responds to this challenge without any adverse effects. However, an abnormal cardiovascular system responds variably (Table 11.3).

FEVER IN A PATIENT WITH HEART DISEASE

Table 11.3: Metabolic changes associated with fever and their significance in cardiac patient

<i>Metabolic change</i>	<i>Clinical significance</i>
<p>Metabolic rate increases by 12% with each degree centigrade increase in temperature</p> <p>Heart rate increases by 15 beats/minute per degree centigrade increase in temperature</p> <p>Increased insensible water loss of 300–500 ml/m²/1 °C/day. Influenced by degree of fever, hyperventilation, humidity, and ambient temperature</p> <p>Electrolyte depletion</p> <p>Hyperventilation early in course of febrile illness with consequent respiratory alkalosis</p>	<p>The net metabolic effect is catabolism. If the need for calories and amino acids is not met, body wasting ensues.</p> <p>Can precipitate heart failure</p> <p>May be responsible for unstable angina or myocardial infarction</p> <p>Expected increase may not occur in patients with sick sinus syndrome, complete heart block and patients receiving calcium or betablocker therapy</p> <p>Expected increase may not occur with typhoid fever, central nervous system infections with intracranial hypertension</p> <p>Hypovolemia may lead to hypotension in diastolic dysfunction, patients on diuretics, vasodilators or nitrates for angina</p> <p>The above drugs may have to be reduced in dosage or withdrawn temporarily</p> <p>Hypokalemia may induce arrhythmias</p> <p>May induce respiratory alkalosis</p> <p>Respiratory alkalosis early and metabolic acidosis late in the clinical course of septic shock</p> <p>Hyperventilation may be mistaken for LVF or acute pulmonary embolism</p>

Pathogenesis

The principal mechanisms of fever in heart disease are infection, tissue necrosis, resolution of a thrombus, hemolysis, or autoimmune phenomenon (Table 11.4).

FEVER IN PATIENTS WITH INFECTIVE ENDOCARDITIS

Fever is the most common presenting symptom and occurs in 85 per cent of patients with infective endocarditis. Chills and sweats are noted in 50 per cent of patients. Though fever is the most common symptom, it may be absent in some patients and some other symptom may dominate the clinical presentation.

Table 11.4: Causes and mechanisms of fever in cardiovascular disease

<i>Causes</i>	<i>Mechanism(s)</i>
Infective endocarditis	Infection Immunological mechanisms Drug induced fever Intravenous lines/phlebitis Metastatic abscesses
Pericarditis	Infection Non-infectious Drug induced (antituberculous)
Rheumatic fever	Immunological response to streptococcal infection
Acute myocardial infarction	Necrosis of myocardium Thrombolytic therapy (STK) Thrombophlebitis at venous access sites Pacemaker lead related phlebitis/infection Post-myocardial infarction Dressler's syndrome
Pulmonary embolism	Thrombus resolution Thrombolytic therapy Septic pulmonary emboli Phlebitis/infection at venous access sites
Aortic dissection	Resorption of hematoma
Systemic hypertension	Aortitis Systemic vasculitis Connective tissue disorders Pheochromocytoma Drug induced (hydralazine) Acute glomerulonephritis Acute pyelonephritis
Left atrial myxoma	Tumour factors Immunologic response
Drug fever	<i>Drugs used in cardiac patients</i> Streptokinase Atropine Dilantin Heparin Hydralazine Meperidine Procainamide Quinidine Salicylates Isoniazid Streptomycin P-aminosalicylic acid

Presenting manifestations of infective endocarditis features

- Fever (85%)
- Weakness, malaise (12–94%)
- Chills, sweats (50%)
- Dyspnea, chest pain, cough (17–42%)
- Skin lesions (25%)
- Headache (50%)
- Onset of hemiparesis (10–20%)
- Personality changes, psychosis (30%)
- Altered sensorium (30%)
- Arthralgia, myalgia (30%)
- Back pain (37%)
- Anorexia, weight loss (15–75%)

Documented fever occurs in 90 per cent of patients but may be absent in a certain subset of patients.

Causes of absence of fever

- Age (elderly patient)
- Congestive heart failure
- Renal failure
- Severe sepsis
- Prior antibiotic therapy
- Fungal endocarditis

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As fever is identified as an essential feature for the diagnosis of infective endocarditis, in the absence of it, the diagnosis may be missed completely. Fever may go unrecognized by some patients but manifests itself as a vague feeling of uneasiness, feverishness, body pain, weakness, loss of appetite or other constitutional symptoms. These symptoms should be considered as fever equivalents.

Any of the above symptoms should arouse the suspicion of elevated temperature and it should be checked.

With appropriate antibiotic therapy, most of the patients with endocarditis become afebrile and

Fever equivalents

Weakness and malaise Arthralgia/body pain Headache Loss of appetite Sweating Insomnia Uneasiness Feverishness
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cultures are negative by the end of first week. Some patients respond more slowly. However, emboli, petechiae, and other peripheral manifestations may occur weeks after starting curative therapy. Heart failure due to valvular damage can occur much later, even years after appropriate therapy. Some patients continue to be febrile in spite of antibiotic therapy; this may be due to inappropriate antibiotic choice but other causes should be checked for:

- Phlebitis, intravenous access sites
- Intramuscular injections
- Drug fever
- Metastatic abscess
- Myocardial or ring abscess
- Rheumatic fever as the cause of fever
- Some other unrelated infection (for example, tuberculosis)
- Superinfection from intravenous catheters. Superinfection of the valve or vegetation by bacteremia or fungemia may occur. It is for this reason that intravenous catheters should be avoided or changed every 48 hours.

FEVER AND POLYARTHRITIS OF RHEUMATIC FEVER

Polyarthritis with fever is the presenting manifestation of acute rheumatic fever, which is the most common form of acquired heart disease in children and adolescents. However a variety of disorders present similarly and require to be differentiated from acute rheumatic fever.

Differential diagnosis

The causes are given in Table 11.5. It is important to consider these conditions, in the differential diagnosis for a patient presenting with polyarthritis and fever (Table 11.6).

RHEUMATIC FEVER

Cardiac involvement dominates the clinical picture in rheumatic fever in children and the arthritis may be less prominent or may be absent. In adults, arthritis is the dominant feature with mild or no carditis. The arthritis is usually abrupt with fever and a third of patients have no recollection of a sore throat. Classic migratory pattern though characteristic is not diagnostic of rheumatic fever and occurs in

FEVER IN A PATIENT WITH HEART DISEASE

Table 11.5: Causes of polyarthritis and fever

<i>Features</i>	<i>Confirmatory</i>
Septic arthritis Bacterial endocarditis Lyme disease Mycobacterial and fungal arthritis Viral arthritis Postinfectious or reactive arthritis Enteric infection Urogenital infection (Reiter's syndrome) Rheumatic fever Inflammatory bowel disease Rheumatoid arthritis and Still's disease Systemic rheumatic illnesses Systemic vasculitis Systemic lupus erythematosus Crystal induced arthritis Gout and pseudogout Mucocutaneous disorders Dermatomyositis Behcet's disease Henoch-Schönlein purpura Kawasaki's disease Erythema nodosum Erythema multiforme Pyoderma gangrenosum Pustular psoriasis Other diseases Cancers Sarcoidosis Familial Mediterranean fever	Synovial fluid and blood culture Blood cultures, Echocardiogram Serologic studies Culture or biopsy Serologic studies Culture or serologic studies Blood culture Clinical findings Clinical findings Biopsy Serologic studies Polarizing microscopy of synovial fluid or tophus Biopsy or clinical findings

other conditions also. Even the rapidly additive large joint involvement is shared by other forms of reactive arthritis. Without aspirin or steroids, fever usually fluctuates without returning to normal for a week or more. Demonstration of recent group A streptococcal infection by serologic studies or throat culture and dramatic response to salicylates supports the diagnosis.

Polyarthritis, fever and bacterial endocarditis

Contrary to expectations, musculoskeletal symptoms are very frequent (44%) in bacterial endocarditis. Arthralgia and low back pain are the most common. Arthritis

Table 11.6: Polyarthritis and fever: diagnostic possibilities derived from discriminating features

<i>Symptom or sign</i>	<i>Diagnostic possibilities</i>
Migratory arthritis	Rheumatic fever Gonococcemia Meningococcemia Viral arthritis Systemic lupus erythematosus Acute leukemia Whipple's disease
Fever preceding arthritis	Bacterial endocarditis Viral arthritis Reactive arthritis Still's disease Lyme disease
Temperature > 40 °C	Bacterial arthritis Still's disease Systemic lupus erythematosus
Pain disproportionately greater than effusion	Rheumatic fever Acute leukemia Familial Mediterranean fever AIDS
Effusion disproportionately greater than pain	Tuberculous arthritis Bacterial endocarditis Inflammatory bowel disease Giant cell arteritis Lyme disease
Morning stiffness	Rheumatoid arthritis Polymyalgia rheumatica Still's disease Any viral and reactive arthritis
Episodic recurrences	Still's disease Crystal induced arthritis Systemic lupus erythematosus Inflammatory bowel disease Whipple's disease Mediterranean fever Lyme disease
Symmetric small joint synovitis	Rheumatoid arthritis Systemic lupus erythematosus Viral arthritis

FEVER IN A PATIENT WITH HEART DISEASE

<i>Symptom or sign</i>	<i>Diagnostic possibilities</i>
Positive test for rheumatoid factor	Rheumatoid arthritis Viral arthritis Tuberculous arthritis Bacterial endocarditis Systemic lupus erythematosus Sarcoidosis Systemic vasculitis
Leukocytosis ($> 15,000/\text{mm}^3$)	Bacterial arthritis Bacterial endocarditis Still's disease Systemic vasculitis Acute leukemia
Leukopenia	Systemic lupus erythematosus Viral arthritis

or joint swelling involving one to three joints occur in 14 per cent of patients. Synovial fluid cultures are usually negative. Rheumatoid factor may be positive in about a third of patients. Arthralgia or arthritis should not be used to differentiate rheumatic fever from infective endocarditis.

APPROACH TO A PATIENT WITH FEVER AND HEART DISEASE

Fever in a patient with structural heart disease

Fever in patients with structural heart defects should always suggest the possibility of infective endocarditis unless proved otherwise. All patients presenting with fever in this background should be investigated for infective endocarditis. This rule should be rigorously applied whether the fever is of one hour, one day, one week or one month duration. The investigation of choice is properly taken blood cultures. It is not a common practice to ask for blood cultures until the fever is prolonged. This is obviously incorrect and blood cultures should be drawn even on the first day of fever in a patient with structural heart defect. The most serious complications like peripheral embolism and heart failure are related to the duration of fever and delayed institution of antibiotic therapy.

Blood cultures

To minimize the risk of contamination, proper method should be followed. Skin contaminants like coagulase negative staphylococci, and diphtheroids may be

misleading. In prosthetic valve endocarditis, even contaminants from the skin may be blood borne and pathogenic.

Techniques:

- Hands should be washed.
- Obtain appropriate media, 2 per cent tincture of iodine, or Betadine, alcohol swabs, tourniquet, disposable plastic syringe and two needles.
- Remove the caps from the culture bottles and using friction, swab the stopper twice with alcohol.
- Select the antecubital site and prepare a circular area with a radius of about 2 inches. Starting at the puncture site, scrub the area with Betadine, using a circular motion with overlapping strokes and working towards the periphery. Repeat the procedure twice. (Use 70 per cent isopropyl alcohol to clean the area for two minutes if the patient is allergic to iodine and Betadine).
- For better recognition of the puncture site, clean the Betadine with alcohol swabs. Follow the same 'clean to dirty' circular motion.
- With a 10 ml syringe, puncture the vein carefully. Avoid touching the needle or prepared skin site. To palpate the vein use a sterile glove.
- Remove the needle from the vein. Avoid touching the needle outside the prepared area.
- Aseptically remove the needle from the syringe and put on the other sterile needle. Transfer not more than 5 ml into each bottle.
- Carefully label the blood culture slip with the date and exact time the culture was drawn. This information is important in interpretation of data later.

Due of low density of most bacteremias, 5 ml samples for adults and 1–5 ml samples for children are commonly recommended. The 1:10 to 1:20 dilution of blood upon inoculation into the culture media usually suffices to reduce intrinsic serum antibacterial activity and reduce the concentration of antibacterial substances to subinhibitory levels.

POST-OPERATIVE FEVER

Fever is common until 5 days after cardiac surgery and is the normal response to tissue disruption and cardiopulmonary bypass. Elevated interleukins may be responsible. Rarely, this normal fever may persist for up to 2 weeks or longer.

Chills are uncommon but may occur. This rise in temperature should be conservatively managed and unnecessary testing should be avoided.

On the other hand, hyperthermia occurring 6–36 hours after open heart operations is usually not due to infection but due to the hypermetabolic state following cardiopulmonary bypass. This hyperthermia is associated with low cardiac output and diminished skin blood flow, which impairs heat loss. This should be considered an emergency and the condition may respond to peritoneal dialysis with cold dialysate. This condition is unrelated to ‘malignant hyperthermia’.

Postoperative wound complications though less common these days, still occur occasionally. Mediastinitis and sternal wound dehiscence are common causes. The risk factors for postoperative wound infection are:

- Prolonged operative time
- Retrosternal hematoma
- Inaccurate sternal closure
- Obesity
- Diabetes
- Bilateral internal mammary dissection
- Male sex (shaving the chest)
- Prolonged postoperative ventilation
- Corticosteroids
- Surgical team and the operative setting (operative time, tissue handling, general ambience)

Unusual fever and malaise, sternal tenderness and persistent severe central chest pain unrelieved by analgesics suggest the possibility of a sternotomy infection. External evidence of inflammation or redness of the skin may be absent. CT scan of the chest may be diagnostic.

FEVER IN PATIENTS WITH CORONARY ARTERY DISEASE

Fever is common in patients with acute myocardial infarction and is related to tissue necrosis. It occurs within 24–48 hours of infarction. The body temperature begins to rise within 4 hours after the onset of infarction. Fever usually subsides by seventh day of infarction. Chills and rigors do not occur and the temperature rarely exceeds 102 °F. Fever may also be related to other factors in acute myocardial infarction, like pericarditis, Dressler’s syndrome or after thrombolysis, especially with streptokinase.

As fever increases the myocardial oxygen demands, it is poorly tolerated by patients with severe coronary artery disease. Significant elevation of temperature may precipitate unstable angina or even acute myocardial infarction. In patients with severe left ventricular dysfunction, congestive heart failure may be precipitated or aggravated. It is for this reason that infection or fever in these patients should be promptly controlled with antipyretics and suitable antibiotics if appropriate. The vasodilatation, loss of water and electrolytes through excessive sweating may induce hypotension, which may be aggravated by nitrates and other antianginal drugs.

The commonest causes of fever in patients hospitalized with acute coronary syndromes are intravenous fluids and indwelling catheters. Adequate antiseptic techniques and changing the lines after 48 hours reduces this complication.

A FEBRILE PATIENT

As pericarditis and infective endocarditis are easily amenable to therapy when recognized early, all patients presenting with fever should be examined to rule out these disorders (Table 11.7). It is not a common clinical habit to look for a pericardial rub or a heart murmur in all patients presenting with fever. Looking for a pericardial rub in a patient with fever involves careful auscultation over the left sternal border at the 3rd and 4th spaces, by pressing the diaphragm during either phase of respiration. One must particularly look for the early diastolic murmur of aortic regurgitation or the soft systolic murmur of mild mitral regurgitation.

Looking for the murmur of aortic regurgitation involves listening with the diaphragm of the stethoscope pressed close to the chest wall, making the patient sit, lean forward, while holding the breath in expiration. Looking for the murmur of mitral regurgitation involves, not only auscultating in the supine position but also doing so in the standing position, to bring out the murmur of mitral valve prolapse.

Congenital bicuspid aortic valve is the commonest congenital anomaly and occurs in 2 per cent of all live births. A bicuspid aortic valve predisposes to infective endocarditis particularly when associated with aortic regurgitation. Takayasu's arteritis is a systemic disease which may present with fever, night sweats, malaise, arthralgias, anorexia and weight loss. These systemic manifestations may precede arterial involvement by months. The disorder most often affects young females. A

FEVER IN A PATIENT WITH HEART DISEASE

Table 11.7: Cardiovascular signs to be looked for in the febrile patient

<i>Disorder</i>	<i>Sign</i>	<i>Maneuver/laboratory test</i>
Pericarditis	Pericardial rub	Left sternal border (3rd, 4th spaces) Diaphragm pressed to chest wall During inspiration (or expiration) Sitting, leaning forward
Infective endocarditis	Early diastolic murmur of AR	Along left sternal border, aortic area, apex Diaphragm pressed to chest wall Sitting, leaning forward, held expiration
	Pansystolic murmur of MR	At the apex Diaphragm of stethoscope Standing for the murmur of MVP
	Ejection click of congenital bicuspid aortic valve	At the aortic area, left sternal border, apex With the diaphragm of stethoscope Mistaken for loud S1 best heard at the aortic area Also mistaken as split S1 best heard at the aortic area
Acute myocarditis	Disproportionate sinus tachycardia Cardiac enlargement Ventricular gallop	Auscultate with the bell lightly applied May be palpable
Takayasu's arteritis	Asymmetry of peripheral pulses Bruit over neck vessels, interscapular area, abdomen	Palpation of all peripheral pulses Look for a bruit over neck, abdomen, interscapular areas
Temporal arteritis	Headache Palpable temporal artery	Palpation of temporal region
Mucocutaneous lymph node syndrome (Kawasaki's disease)	Cervical adenopathy Erythema of skin Erythema of mucous membranes Desquamation of skin and finger tips Pericardial rub due to pericarditis Signs of myocarditis Signs of myocardial ischemia or infarction	ECG evidence of myocardial ischemia or infarction Coronary artery aneurysms made out by echocardiography and coronary angiogram

<i>Disorder</i>	<i>Sign</i>	<i>Maneuver/laboratory test</i>
Recurrent pulmonary emboli	Fever may dominate the clinical picture Dyspnea may not be prominent Sinus tachycardia is common	Ventilation perfusion lung scan Pulmonary angiogram
Phlebitis	Intravenous line sites Redness, pain, swelling	

decrease or absence of arterial pulses, bruit over the neck, chest, or abdomen, or blood pressure difference between the upper and lower limbs in any young woman presenting with fever of obscure origin is suggestive of Takayasu's arteritis. Kawasaki's disease is an acute febrile multisystem disease in children. The affected children have non-suppurative cervical adenitis failing to respond to antibiotics, edema, conjunctival congestion, erythema, of oral cavity, lips, and palms, and desquamation of the skin and finger tips. Coronary vasculitis is the cause for myocardial ischemia and infarction. Echocardiography and coronary angiography reveal coronary artery aneurysms. Recurrent minute pulmonary emboli may present with fever as the dominant manifestation and the commonly expected dyspnea may not be impressive. Ventilation perfusion lung scan and pulmonary angiogram may be confirmatory.

PRACTICE IMPLICATIONS

- In any patient with fever, whether it is the first hour, first day or first week, look for a heart murmur. If you don't do this, an opportunity to detect infective endocarditis at an early stage is missed.
- Looking for a heart murmur means truly looking for it by careful auscultation at rest and with various maneuvers.
- Surprisingly, a significant number of patients with infective endocarditis fail to mention fever unless queried by the doctor. This is because the fever is either low-grade or is attenuated by antibiotics.
- Arthralgias and even arthritis does not distinguish rheumatic fever from infective endocarditis.
- Musculoskeletal symptoms are common in infective endocarditis.

FEVER IN A PATIENT WITH HEART DISEASE

- In a patient with structural heart disease, any fever has to be viewed with suspicion to rule out infective endocarditis from the beginning.
- Nowadays, the most common presenting feature of infective endocarditis is recurrent fever due to indiscriminate use of antibiotics. All patients with valvular and congenital heart disease should be educated regarding this.

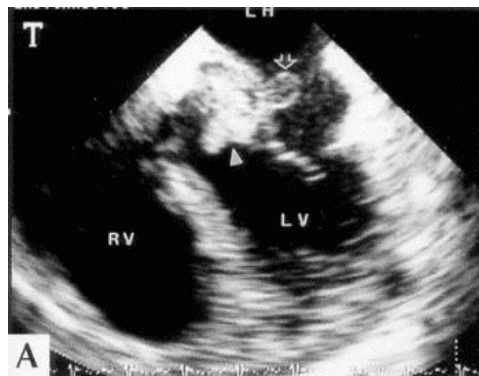


Fig. 11.1: Vegetation on aortic valve as seen in transesophageal echocardiography

12 The Arterial Pulse

The arterial pulse reflects the performance of the left ventricle and the response of the vascular system to left ventricular ejection. The palpable arterial pulse is the pressure pulse transmitted along the arterial system as with each systole, blood is ejected by the left ventricle into the proximal aorta. The normal arterial pulse consists of an ascending limb, the peak, and the descending limb. The pulse wave is transmitted along the aorta to the periphery at a speed of 5 m/sec but the intraluminal blood travels much slower, at only 40–50 cm/sec. The main determinants of the morphology of arterial pulse are outlined in the Table 12.1.

With a normal stroke volume, the normal upstroke of the arterial pulse occurs. When the stroke volume increases, the upstroke is sharper and the peak is higher, resulting in a larger pulse volume. The reduction in the stroke volume has the opposite influence. As the velocity of ejection increases, the upstroke is sharper, and the peak is reached earlier. When the velocity of left ventricular ejection decreases with reduced contractility, the upstroke is less sharp and the peak is

Table 12.1: Determinants of arterial pulse

Left ventricle	Stroke volume Left ventricular contractility Velocity of left ventricular ejection
Aortic valve	Normal Stenotic Regurgitation Combined stenosis and regurgitation
Arterial system	Compliance or distensibility Peripheral vascular resistance Aortic run off

THE ARTERIAL PULSE

reached later. With a regurgitant aortic valve, the descending limb of the arterial pulse is steeper and the attendant increase in stroke volume results in a sharper upstroke with a higher peak, giving rise to the collapsing pulse of aortic regurgitation. In aortic stenosis, the arterial system is filled slowly, resulting in the slow rising upstroke and a delayed peak.

NORMAL ARTERIAL PULSE

The normal arterial pulse consists of the upstroke, the peak and the descending limb. The upstroke comes out with the first sound and the peak is reached well before the second heart sound. In the central arterial pulse, the peak of the arterial pulse consists of an initial percussion wave, and a smaller and later-occurring tidal wave. The percussion wave is due to the initial left ventricular ejection, and the tidal wave is due to aortic recoil or a reflected wave from the periphery.

The normal arterial pulse consists of a rapid upstroke (percussion wave, P) and a second wave in systole, called the tidal (T) wave. The end of systole is indicated by the sharp dicrotic notch, which is followed by a dicrotic wave. These wave forms are better recorded in the central arteries. The dicrotic notch and dicrotic wave are not clearly discernable towards the periphery.

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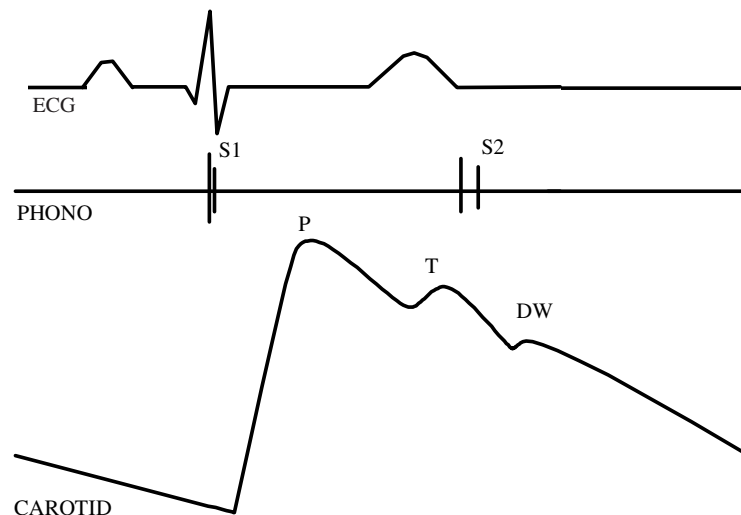


Fig. 12.1: Normal arterial pulse

P: Percussion wave, T: Tidal wave, DW: Dicrotic wave

Technique of palpating the pulse

The carotid artery is the most suitable for evaluating the character of the arterial pulse, because it is near the aortic valve. If the carotid artery is not available, the brachial artery is the next choice. A more peripheral pulse like radial artery is suitable when attempting to appreciate a collapsing pulse or a dicrotic pulse since these waves are exaggerated as the pulse wave is transmitted to the periphery. The paradoxical pulse is best appreciated at the femoral artery as the patients with cardiac tamponade usually have low pulse volume, and severely elevated jugular venous pressure, which interferes with palpation of the carotid pulse. To palpate the carotid pulse, the patient should be lying supine, with the neck slightly turned to the side of palpation to relax the sternomastoid muscle. The carotid artery is felt by using two or three fingers (the middle three fingers), along the lateral border of the trachea. By simultaneous auscultation and palpation, the upstroke of the carotid pulse appears as an abrupt outward movement along with the first heart sound and the peak is reached well before the second heart sound. A systematic approach to the arterial system requires looking for the following features.

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Evaluation

- Rate and rhythm
- Volume, tension
- Character
- Vessel wall
- Peripheral pulses
- Grade the palpability
- Brachiofemoral and brachio-brachial delay
- Bruit over the artery
- Palpation of the abdominal aorta
- Ocular fundi
- Allen's test

Rate and rhythm: The arterial pulse is often the inaugural part of the physical examination, and one often forms the initial impressions regarding the patient's illness at this stage. The pulse rate should be counted for at least half a minute to be reliable. The normal range of pulse rate is 60–90/min. Pulse rates above 140/min cannot be reliably counted. In infants, the rates are higher, and can reach 160/min.

THE ARTERIAL PULSE

The **causes of rapid regular pulse** (more than 90/minute) could be:

- Sinus tachycardia
- Supraventricular tachycardia
- Paroxysmal atrial tachycardia
- Junction tachycardia
- Atrial tachycardia with fixed block
- Atrial flutter with fixed block
- Ventricular tachycardia

The **causes of sinus tachycardia** could be:

- Anxiety
- Visit to the doctor
 - Long waiting period
 - Doctor's personality and attitude
- Emotion
- Fever
- Septicemia with or without fever
- Pregnancy
- Vasodilator drugs
- Soon after consuming food or beverages.

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The **causes of slow pulse** (less than 60/minute) could be:

- Sinus bradycardia
- Complete heart block
- Heart block of 2:1 or 3:1
- Bigeminal rhythm with impalpable premature beat
- Pulsus alternans with the weak beat impalpable

The **causes of sinus bradycardia** could be:

Physiological

Normal variant
Athletes
Neonates
During sleep

Vagal overactivity

Vasovagal episodes
Acute inferior myocardial infarction

Any visceral pain
 Sick sinus syndrome
 Intracranial hypertension
 Myxedema
 Hypothermia
 Mediastinal tumours
 Obstructive jaundice
 Convalescence from infections
 Depression
 During reperfusion after thrombolytic therapy

Drugs

Betablockers
 Digoxin
 Some calcium blockers (verapamil, diltiazem)
 Clonidine
 Lithium
 Amiodarone

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One must consider whether the rate is appropriate or inappropriate for the clinical circumstance. For example, a patient with fever should have sinus tachycardia. If the expected tachycardia fails to occur in a patient with fever one must consider various possibilities.

The **causes of normal or slow pulse rate with fever** (relative bradycardia) could be:

- Typhoid fever
- Viral infections
- Hemorrhagic fevers
- Lassa fever
- Lymphocytic choriomeningitis
- Intracranial infection with intracranial hypertension
- Meningitis
- Brain abscess
- Encephalitis
- Fever in a patient with
 - Bradyarrhythmia
 - Drug induced bradycardia

THE ARTERIAL PULSE

The **causes of rapid irregular pulse** could be:

- Atrial fibrillation
- Atrial flutter with varying block
- Atrial tachycardia with varying block
- Frequent atrial and ventricular ectopy

An irregularly irregular pulse suggests the possibility of atrial fibrillation. If the irregularity is predictable, as in frequent premature ventricular contractions, it is called a regularly irregular pulse. Slight respiratory alteration in pulse rate is common in children and is called sinus arrhythmia. It is of two forms, the respiratory form where the rate increases during inspiration and decreases during expiration, and the non-respiratory sinus arrhythmia characterized by phasic variation in heart rates unrelated to respiratory cycle, and is often suggestive of digitoxicity.

The importance of the pulse rate is increasingly recognized by the recent information that the pulse rate is a major correlate of blood pressure. Rapid heart rates may predict the development of sustained hypertension in subjects with normal or borderline elevated blood pressures. Sinus tachycardia is associated with increased risk of cardiovascular death. Also sinus tachycardia is the single most important long term prognostic factor following acute myocardial infarction.

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Pulse volume: The amplitude of excursion of the pulse is used to assess the volume of pulse and generally correlates with the stroke volume. In elderly patients with rigid atherosclerotic aorta and in systemic hypertension, the pulse volume is high due to non-distensible arterial system (Table 12.2). In these settings, the pulse volume is not a true reflector of stroke volume.

Table 12.2: Causes and mechanisms of high pulse volume

<i>Cause</i>	<i>Mechanism</i>
Elderly	Atherosclerotic non-distensible arterial system
Emotional excitability, anxiety	↑Stroke volume
High cardiac output states like anemia, thyrotoxicosis	↑Stroke volume
Conditions with aortic run off	Low diastolic pressure
	Low diastolic pressure
	↑Stroke volume
Hyperkinetic heart syndrome	↑Stroke volume
	↓Systemic resistance
Systemic hypertension	Non-distensible arterial system

In a typical patient with systemic hypertension, the rise in systolic pressure is not in proportion to the elevation in diastolic pressure.

The **causes of low pulse volume** could be:

- Shock
- Low cardiac output
- Myocardial disease
- Valvular disease
- Pericardial disease
- Acute hypertension as in eclampsia of pregnancy
- Hypovolemia

Character of the pulse

Normal arterial pulse: The normal arterial pulse consists of an abrupt upstroke coming up with the first sound, and a peak which occurs well before the second sound. This is best appreciated in the carotid pulse by simultaneous auscultation and palpation.

The best way to become proficient in the technique of pulse evaluation is to practice this in otherwise normal people or by palpating one's own carotid.

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Collapsing pulse or water hammer pulse: The collapsing pulse refers to the rapid descent in the arterial pulse and is accompanied by a sharp upstroke and a high volume. The collapsing pulse occurs in all situations where there is run off of blood from the aorta or the arterial system. Aortic regurgitation is the most important cause of a collapsing pulse. The collapse is related to the back flow into left ventricle, and the reflex vasodilatation mediated by the carotid baroreceptors secondary to the large stroke volume. In other words, the arterial system in aortic regurgitation behaves as if it is open at both ends in diastole, unlike the normal, which is open only at the arteriolar end of the circulation. This sign is best appreciated at the radial pulse, with the palmar side of the wrist held in the examiner's hand, and the arm elevated above the shoulder. This may be related to the artery being more in line with the central aorta allowing direct systolic ejection and diastolic backward flow. All the peripheral signs of aortic regurgitation are related to this collapsing pulse.

It is not essential to elicit all these signs in a patient with aortic regurgitation. Hill's sign is particularly important, because it allows estimation of severity of

THE ARTERIAL PULSE

aortic regurgitation and correlates well with angiographic severity of aortic regurgitation. The collapsing pulse is not specific for aortic regurgitation and occurs in a variety of conditions.

Table 12.3: Peripheral signs of aortic regurgitation

<i>Sign</i>	<i>Description</i>	<i>Mechanism/Significance</i>
Hill's sign	Systolic BP in lower limb higher by more than 20 mmHg	Phenomenon of recruitment of reflected waves < 20: Trivial AR/Normal 20–40: Mild AR 40–60: Moderate AR > 60: Severe AR
Duroziez's double murmur	To and fro murmur over the femoral artery	Forward murmur due to increased stroke volume Backward murmur due to arterial recoil and backflow Always indicates severe AR Very large SV, low diastolic pressure
Pistol shot femorals	Audible sound during systole coinciding with upstroke of FA	Moderate to severe AR
de Musset's sign	To and fro motion of the head synchronous with systole and diastole	Large stroke volume Severe AR
Quincke's sign	Advancing and retreating flush with each cardiac cycle by light pressure over nail bed, mucous membranes of mouth	Arteriolar dilatation Severe AR
Traube's sign	Booming systolic and diastolic sounds over FA	As for Duroziez's murmur
Muller's sign	Pulsations in the uvula	As above
Gerhardt's sign	Pulsations in the enlarged spleen	As above
Rosenbach's sign	Pulsations in the liver	As above
Ladolfi's sign	Change in pupil size with each cardiac cycle	
Pulsations of retinal vessels	Appearance and disappearance of blood column with cardiac cycle	Severe AR

The **causes of a collapsing pulse** could be:

Hyperkinetic circulatory states

- Pregnancy
- Fever
- Anemia
- Thyrotoxicosis
- Beriberi
- Paget's disease of bone
- Hyperkinetic heart syndrome
- Cirrhosis of liver
- Drug induced vasodilatation

Conditions with aortic run-off

- Aortic regurgitation
- Patent ductus arteriosus
- Aortopulmonary window
- Rupture of sinuses of Valsalva into right heart chambers
- Arteriovenous fistula
- Systemic to pulmonary artery shunt in cyanotic heart disease with reduced pulmonary flow

In all the above states, a collapsing pulse generally means a severe lesion.

Slow rising pulse of aortic stenosis (anacrotic pulse, pulsus tardus)

In all forms of fixed obstruction to left ventricular outflow, the upstroke of the arterial pulse is slow, and the peak is delayed nearer to the second sound. This is best appreciated by simultaneous auscultation of the heart and palpation of the carotid pulse.

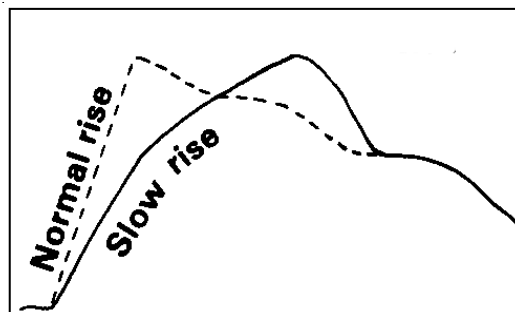


Fig. 12.2: Slow rising pulse in aortic stenosis

Slow rising pulse: The correlate of this type of pulse in aortic stenosis is, a gradient of at least 70 mmHg across the aortic valve or a severe aortic stenosis (Fig. 12.2). At extremes of age, this sign may be absent, as in children with rapid velocity of circulation and elderly with non-distensible arterial system. For the same reason, elderly patients with significant AS may have coexistent systolic hypertension. These patients belong to a subset with not only a dynamic left ventricle but also a markedly non-compliant arterial system. The delay and the slow rise may not be appreciable once heart failure sets in, when only the low volume pulse may be appreciated. If the pulse is normal in the setting of aortic stenosis, one should consider various possibilities.

Pulsus parvus et tardus (slow rising small pulse): This is typically present in a patient with severe aortic stenosis. The upstroke of the pulse is very slow-rising and the pulse volume tends to be low due to elevated systemic vascular resistance. In dynamic obstruction, as in hypertrophic obstructive cardiomyopathy, despite severe left ventricular outflow tract obstruction, the pulse tends to be brisk.

The causes of normal arterial pulse with aortic stenosis could be:

- Mild aortic stenosis or conditions simulating aortic stenosis (for example MR)
- Associated AR
- Hypertrophic obstructive cardiomyopathy
- Supraaortic aortic stenosis (normal right arm pulse)
- Associated coarctation of aorta
- Associated mitral stenosis
- Extremes of age: children and elderly

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In severe aortic stenosis, the slow rising pulse with a delayed peak, the long ejection systolic murmur with late peaking and the sustained apical impulse go hand in hand.

Bisferiens pulse: In the bisferiens pulse both the percussion and tidal waves are appreciable; it occurs in a variety of conditions.

- Severe aortic regurgitation (Fig. 12.4)
- Aortic stenosis and regurgitation
- Hypertrophic obstructive cardiomyopathy (Fig. 12.3)
- Hyperkinetic circulatory states
- Muscular exercise

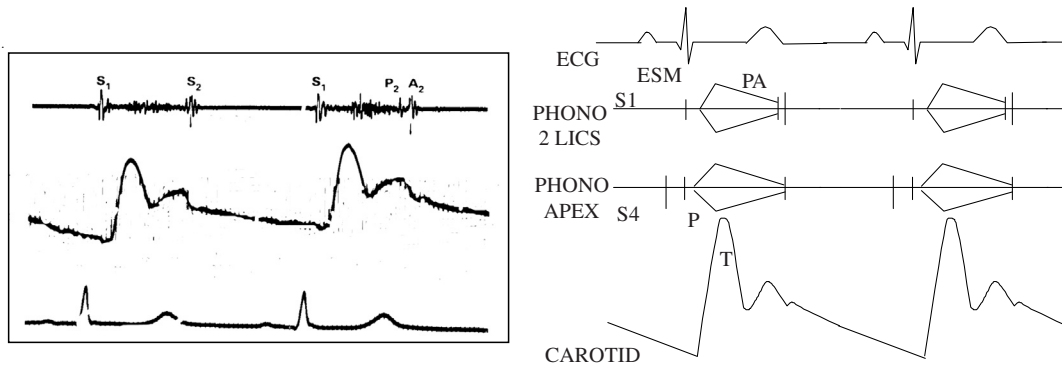


Fig. 12.3: Bisferiens pulse in HOCM

Bisferiens pulse in hypertrophic obstructive cardiomyopathy (HOCM): Typically, in HOCM, the pulse is bisferiens, that is, both the percussion and tidal waves are well appreciated. Characteristically, the percussion wave is prominent in HOCM.

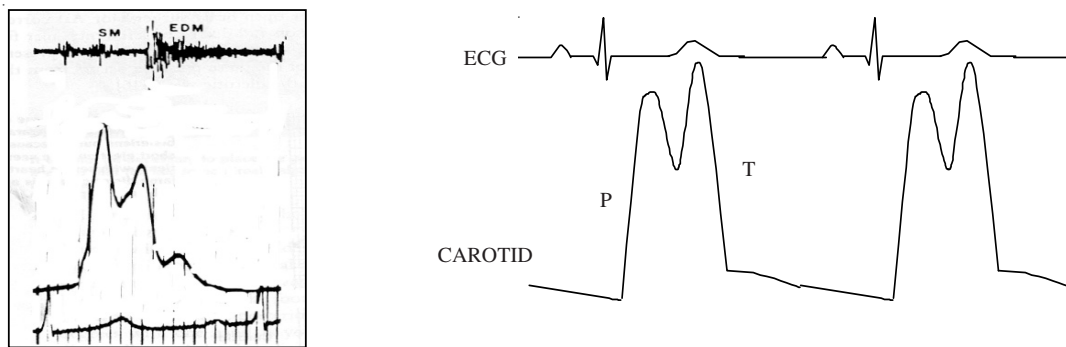


Fig. 12.4: Bisferiens pulse in severe AR

Bisferiens pulse in severe aortic regurgitation: In severe AR, the character of the pulse is bisferiens. Unlike in HOCM, in AR both the tidal and percussion waves are prominent. More often, the bisferiens pulse is seen, when there is associated mild aortic stenosis.

Mechanisms of bisferiens pulse: Normally the percussion wave is felt but not the tidal wave. The tidal wave is due to the elastic recoil of the aorta and the reflection wave from the periphery. In all situations where the initial percussion wave is exaggerated, the tidal wave is also prominent. This mechanism is applicable in severe aortic regurgitation and other hyperkinetic circulatory states. In combined aortic stenosis and aortic regurgitation, the stenotic component permits a jet. Lateral to the velocity jet, there occurs a fall in pressure (Bernoulli phenomenon). This results in a dip or inward movement in the pulse with a secondary outward movement or tidal wave. In hypertrophic cardiomyopathy, the initial part of left ventricular ejection is normal or more rapid than normal, resulting in a rapid

upstroke. As obstruction to outflow starts later in systole, due to thickening of interventricular septum and systolic anterior motion of the anterior leaflet of the mitral valve, a sudden interruption to left ventricular ejection occurs resulting in a dip in the pressure pulse followed by the slow rising pulse wave like ordinary aortic stenosis. In effect, the arterial pulse of idiopathic hypertrophic subaortic stenosis behaves partly like aortic regurgitation (the initial sharp component) and aortic stenosis (the later slow rising wave).

Technique of palpating for bisferiens pulse: This is usually best appreciated on the carotid but the prominent systolic thrill over the carotid (the carotid shudder) may mask the features. A more distal pulse like the brachial or the radial may be suitable. One should use a graduated pressure to be able to appreciate the two waves. Alternatively completely obliterate the pulse, and gradually release it, and at one point the two waves are appreciable.

Dicrotic pulse: The dicrotic pulse occurs when the dicrotic wave is exaggerated. The dicrotic wave is possibly related to the reflection wave from the periphery. When the reflection wave travels slowly, and gets added to the original wave, an additional dicrotic wave is appreciable (Fig. 12.6). On the other hand, when the reflection wave travels rapidly and meets the original wave well in advance, it is lost in it.

When the arterial system is rigid and non-distensible as in systemic hypertension, the dicrotic pulse is never present (Fig. 12.5). The dicrotic pulse is commonly seen in low cardiac output states like cardiomyopathy, myocarditis, cardiac tamponade or after aortic valve replacement for aortic regurgitation. In the above states with a low cardiac output and the underfilled arterial system, the reflection wave travels slowly to be appreciated as an additional wave. Dicrotic pulse is commonly seen in typhoid fever and is possibly related to the circulating vasculotoxins.

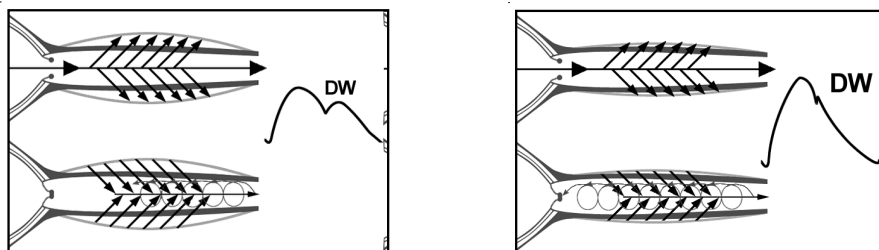


Fig. 12.5: Pulse wave and reflected wave in elastic aorta (left) and stiff aorta (right)

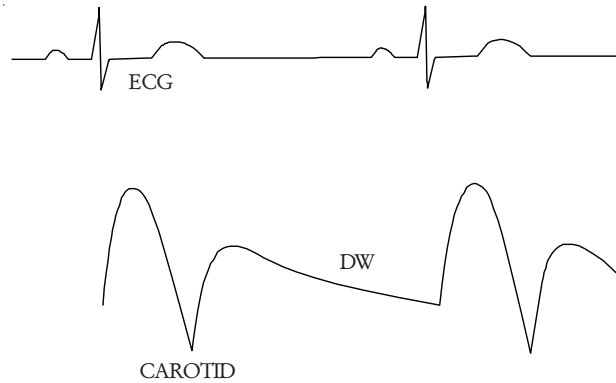


Fig. 12.6: Dicrotic wave (DW)

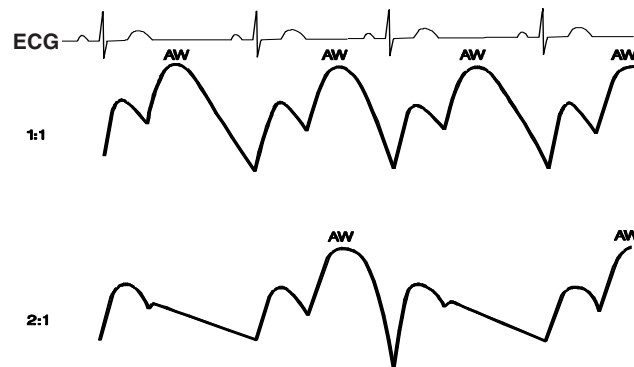


Fig. 12.7: Augmented wave (AW) with IABP usage

The **causes of a dicrotic pulse** are:

- Typhoid fever or any fever with vasodilatation
- Any condition with low cardiac output
- Cardiomyopathy
- Myocarditis
- Cardiac tamponade
- Hypovolemia
- During intra-aortic balloon pumping

The dicrotic pulse is distinguished from the bisferiens pulse by the second wave occurring after the second heart sound. Simultaneous auscultation is helpful. The bisferiens pulse is generally best felt over the carotid but the dicrotic pulse is most often appreciated over the radial pulse. It is better appreciated during

inspiration or inhalation of amyl nitrite. The dicrotic pulse is unusual when the systolic pressure exceeds 130 mmHg, or in a person beyond 50 years of age.

Dicrotic pulse during intra-aortic balloon pumping (IABP): With the increasing use of IABP in various clinical settings, the dicrotic pulse is more often recorded (Fig. 12.7). In this setting, the prominent dicrotic wave is the augmented wave resulting from diastolic flow occlusion in the descending aorta due to the inflation of the balloon.

Bigeminal pulse: The bigeminal pulse is due to the bigeminal rhythm. Alternating beats are strong and weak (Fig. 12.8). However, unlike pulsus alternans, these beats do not occur regularly. This is commonly seen with ventricular bigeminy. The weak beat is prematurely close to the previous normal beat and the weak beat is followed by a long pause. The stronger beat correlates with preceding longer diastole due to post-extrasystolic pause resulting in greater left ventricular end diastolic volume. The bigeminal pulse is often accompanied by auscultatory bigeminy. Sometimes the weak contraction may be able to close the mitral valve producing the first heart sound but unable to open the aortic valve to produce the second sound. In the latter case it may be mistaken for a third heart sound. When the premature beat is very low in volume or is not felt, the pulse rate counted may be half of the heart rate and a mistaken diagnosis of bradycardia is often made. The premature beat is often accompanied by a cannon wave in the neck veins.

Post-extrasystolic pulse: Normally, the post-extrasystolic pulse shows an increase in volume, due to the long pause and more diastolic filling. More importantly, the extrasystolic potentiation of ventricular contraction also contributes to this. This normal pattern is seen in all forms of fixed obstruction to the left ventricular outflow. In hypertrophic obstructive cardiomyopathy, the extrasystolic potentiation increases the obstruction, and decreases the pulse volume of the post-extrasystolic pulse (Fig. 12.9). Lack of rise of post-extrasystolic beat by 10 mm Hg or actual fall in pulse is called *Brockenbrough sign* and is the surest sign of a dynamic obstruction to left ventricular outflow (Table 12.4). Even failure of the pulse volume to rise should be considered a positive sign but this may also be seen in severe ventricular dysfunction or constrictive pericarditis.

Pulsus alternans: This is characterized by a regular sinus rhythm with alternate beats strong and weak due to alternation in contraction of the heart (Fig. 12.10).

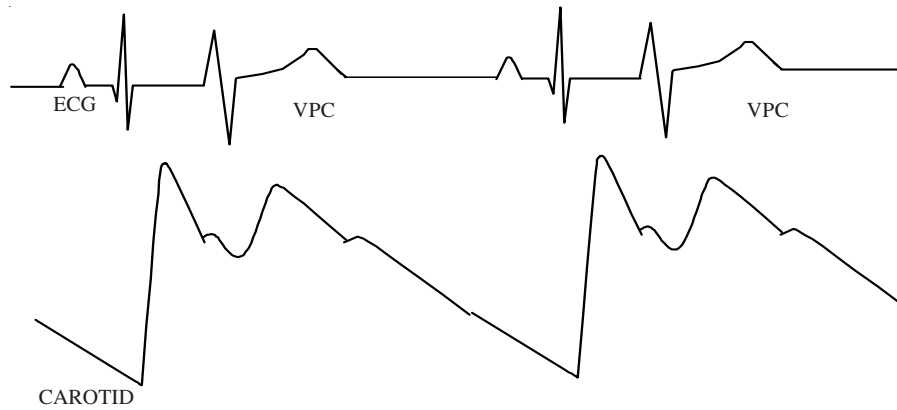


Fig. 12.8: Bigeminal pulse

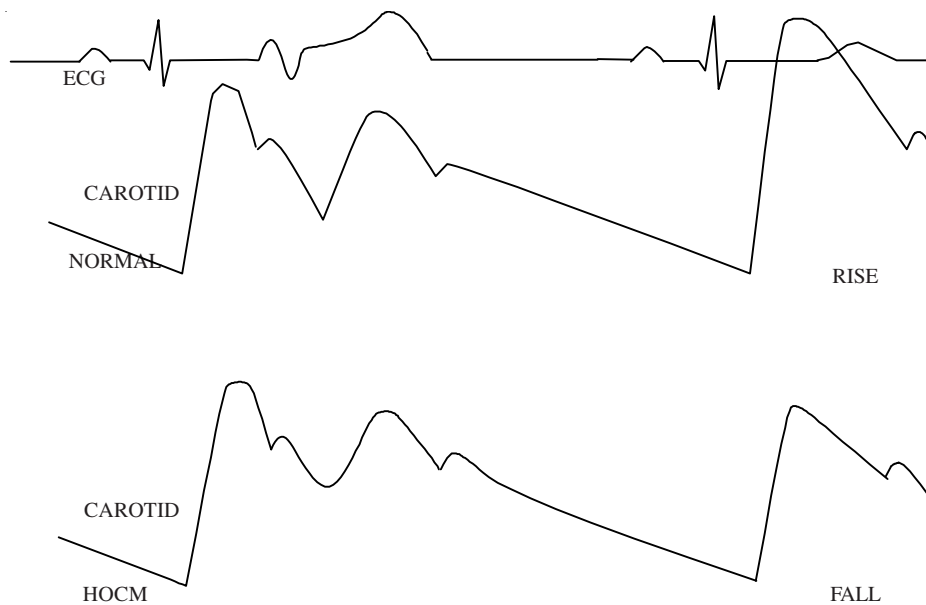


Fig. 12.9: Postectopic behaviour of pulse

Normal compared to HOCM. Note the absence of post-extrasystolic potentiation in HOCM

The equivalent of this by sphygmomanometry is alternation of the intensity of Korotkoff sounds.

It was first described by Traube in 1872. It is often a sign of severe myocardial depression and is related to more and less number of contractile elements participating in each contraction alternately. Pulsus alternans is most commonly seen in aortic stenosis with heart failure but also occurs in any condition with

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severe ventricular dysfunction. Alternans can be total, when left ventricular systolic pressure generated is less than the aortic diastolic pressure when the left ventricle fails to open the aortic valve. When it occurs on both sides of the heart (right and left ventricles), it is called *concordant*. When it involves only the right or the left ventricle, it is called *discordant*. In aortic stenosis, the strong and weak beats are appreciated as variation in the intensity of the murmur alternately. When the weak beat is too weak to be palpable, only auscultatory alternans is appreciable. Aortic regurgitation, systemic hypertension and reducing the venous return by head tilting or nitroglycerine usually exaggerate pulsus alternans and assist in its detection. Premature ventricular contractions, rapid atrial pacing, inferior vena caval occlusion, myocardial ischemia and intracoronary injection of contrast during coronary arteriography are known to induce alternans.

Table 12.4: Causes of positive Brockenbrough sign

<i>Cause</i>	<i>Mechanism</i>
Hypertrophic obstructive cardiomyopathy	Post-extrasystolic potentiation of dynamic LV outflow obstruction
Constrictive pericarditis	Failure to fill the ventricle more even after longer diastole due to constriction
Severe ventricular dysfunction due to any cause	Failure to augment contraction in spite of more preload
Extremely severe aortic stenosis at any site	Same as above

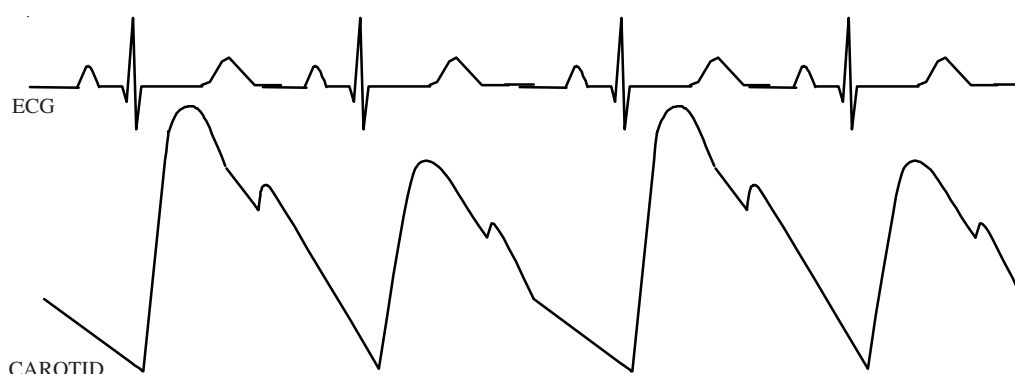


Fig. 12.10: Pulsus alternans

Definitions

1. Pulsus alternans: Regular alternation of strong and weak pulse in the absence of respiratory or cycle length alteration.
2. Total alternans: When the weak contraction fails to open the aortic valve, alternans pulsus may be absent.
3. Independent alternans: Pure alternans of either right or left ventricles alone.
4. Concordant alternans: Simultaneous alternans of right and left ventricles.
5. Discordant alternans: Alternating alternans of right and left ventricles.
6. Compound alternans: Further alternation of the weaker beats superimposed on the routine alternans.

Pulsus alternans is easier to detect by sphygmomanometer (Fig. 12.11) but when the aortic pressure shows alternans by more than 20 mmHg, it can be detected by palpation of the peripheral pulse. Pulsus alternans is usually accompanied by alternation in the intensity of Korotkoff sounds. Electrical alternans is an independent phenomenon and has no relationship to pulsus alternans.

The **causes of pulsus alternans** could be:

- Severe aortic stenosis
- Severe pulmonic stenosis
- Dilated cardiomyopathy
- Myocarditis
- Severe aortic regurgitation with left ventricular failure especially after aortic valve replacement

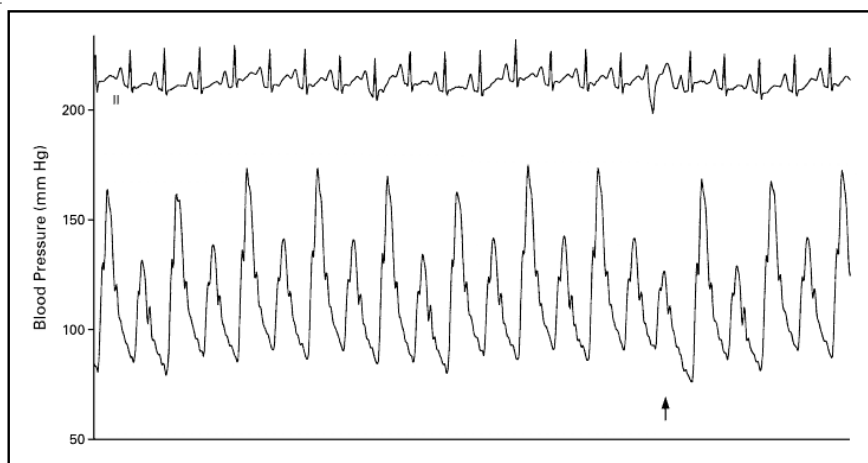


Fig. 12.11: Pulsus alternans as noted in blood pressure

- Acute pulmonary embolism
- Severe coronary artery disease
- Any cause for ventricular dysfunction
- Briefly during or after supraventricular tachycardia
- Severe systemic hypertension
- Transient right ventricular outflow occlusion during balloon dilatation of pulmonary stenosis
- Normal hearts (rare)

Pulsus alternans is accompanied by alternating intensity of heart sounds and or S3 gallop.

The precise mechanism of this phenomenon is not known. It may primarily be due to decreased myocardial contractility of alternate beats with relatively less effect produced by changes in preload, afterload or diastolic relaxation. Decreased contractility is attributed to deletion of a number of myocardial elements with alternate beats. Intracellular calcium cycling involving the sarcoplasmic reticulum leading to localized electromechanical dissociation. Alterations in diastolic volume or relaxation may also play a role.

Pulsus alternans is best appreciated by light pressure and the patient holding the breath in mid expiration. This limits respiratory influence on pulse volume. Pulsus alternans is augmented by maneuvers, which decrease venous return, such as standing or administration of nitroglycerine.

Compound pulsus alternans: Sometimes, an additional alternation involving only the weak beats can occur in association with the usual alternation of weak and strong beats, and is known as compound pulsus alternans.

Pulsus alternans may disappear with improvement in ventricular function or administration of digitalis. It may also disappear with progressive deterioration of ventricular function. It is for this reason, that a new appearance or disappearance of this sign should be viewed with concern.

Pulsus paradoxus: Normally there is an inspiratory fall and expiratory rise in systolic pressure related to the alteration in the stroke volume of the left ventricle. In pulsus paradoxus there is an exaggerated fall in systolic arterial pressure with inspiration. The normal inspiratory fall in systolic pressure is usually less than 8 mmHg. Any exaggeration from this is called pulsus paradoxus (Fig. 12.12).

It can occur in any condition with an exaggerated respiratory act, as in a severe attack of asthma or in any other form of obstruction to the airways.

The **causes of pulsus paradoxus** could be:

Normal

- Pregnancy
- Exaggerated inspiration voluntarily (student paradoxus)
- Extreme obesity

Severe obstruction to airways

- Acute severe bronchial asthma
- Severe emphysema
- Upper airway obstruction

Pericardial tamponade

- Tense pericardial effusion
- Severe constrictive pericarditis
- Effusive constrictive pericarditis
- Hypovolemic shock
- Massive pulmonary embolism

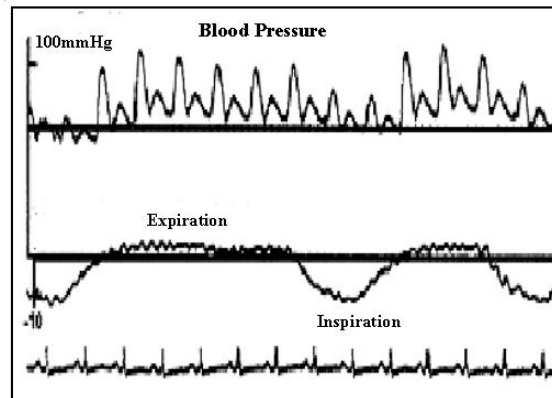


Fig. 12.12: Pulsus paradoxus: Note the exaggerated fall in blood pressure with inspiration

Table 12.5: Cause and mechanism of absence of paradoxus in cardiac tamponade

Cause	Mechanism
Atrial septal defect	Equal filling of ventricles in either phase of respiration
Ventricular septal defect	Free communication between ventricles prevents differential filling
Aortic regurgitation	Filling of LV is maintained irrespective of respiration

In cardiac tamponade, the right and left ventricles are equally compressed by the tense pericardial effusion. Normally the inspiratory increase in venous return is accommodated by the easily distensible right ventricle. In cardiac tamponade, the increased venous return to the RV cannot be accommodated as in normal situations and the RV distension occurs at the expense of left ventricle resulting in reduced left ventricle end diastolic volume and stroke volume. The evidence can be seen in the echocardiogram as increase in RV dimension, displacement of interventricular septum toward left ventricle and reduction in the left ventricle dimension during inspiration. The opposite of all this occurs in expiration.

The femoral pulse is most convenient for appreciating a paradoxical pulse because most patients with paradoxical pulse (cardiac tamponade, constrictive pericarditis and obstructive pulmonary disease) have neck edema, and the carotid pulse is therefore difficult to palpate. In obstructive pulmonary disease, the accessory activity of the neck muscles interferes with carotid palpation. The patient should be breathing normally, and it is a wrong practice to ask the patient to take a deep breath. Otherwise normal people can have a paradoxus with deep breathing. To measure the pulsus paradoxus the sphygmomanometer cuff is inflated to above the level of systolic pressure and is deflated very slowly (2 mmHg/beat). The level at which the first Korotkoff sounds are heard is marked. Initially the sounds are intermittently heard during expiration, and are inaudible during inspiration. As the cuff is deflated slowly, the sounds are heard continuously at one point in both phases of respiration. The difference between the point of intermittent audibility of sounds to the point of continuous audibility gives the degree of paradoxus. By definition, it should be above 10 mmHg. Any value above 15 mmHg is called *significant paradoxus*. When the paradoxus is extreme, the pulse may disappear only to reappear during expiration, in which case, a mistaken diagnosis of atrial fibrillation, or some other arrhythmia is often made. However on auscultation, the heart is regular. This seeming irregularity of the arterial pulse but regular beating of heart sounds, was considered paradoxical by Kussmaul, who originally described this pulse and named it thus. The principal determinant of pulsus paradoxus is reduced filling of the left ventricle during inspiration in relation to the right ventricle. Any condition which permits equal filling of both ventricles (shunt lesions), or more filling of left ventricle (aortic regurgitation) prevents paradoxus from occurring (Table 12.5).

Table 12.6: Causes and mechanisms of reversed pulsus paradoxus

<i>Cause</i>	<i>Mechanism</i>
Positive pressure breathing with artificial ventilators Isorhythmic AV dissociation	Intrathoracic pressure is higher in inspiration and lower in expiration The atrial activity precedes ventricular activity during inspiration and follows it during expiration. The atrial contribution during inspiration increases stroke volume and the lack of it during expiration decreases it
Hypertrophic obstructive cardiomyopathy	Mechanism not known

Reversed pulsus paradoxus: Inspiratory increase and an expiratory decrease may occur during positive pressure breathing of artificial ventilation, as the intrathoracic pressure is high during inspiration and low during expiration (the reverse of normal). In this setting if cardiac tamponade occurs an expiratory fall is seen and this is the reverse of routine pulsus paradoxus. Similar manifestation may be seen during isorhythmic AV dissociation when the P wave precedes the QRS during inspiration and marches into the QRS during expiration. Reversed pulsus paradoxus is also described in hypertrophic cardiomyopathy but the mechanism is not clear (Table 12.6).

The peripheral pulses

Palpation of the peripheral pulses is helpful in the diagnosis of peripheral vascular disease, coarctation of the aorta, aortic dissection and embolic manifestations of atrial fibrillation or infective endocarditis, Takayasu's arteritis and some vasculitis syndromes. The following system of expressing the results in a tabular form is helpful. All the accessible arteries should be palpated bilaterally including the abdominal aorta. Auscultation for a bruit should be looked for over the femorals, ilio-femorals, abdominal aorta, renal arteries, celiac trunk or superior mesenteric artery and the carotids.

Recording of normal peripheral pulses

Artery	Carotid	Brachial	Radial	Femoral	Popliteal	Posterior tibial	Dorsalis pedis
Right	+++	+++	+++	+++	+++	+++	+++
Left	+++	+++	+++	+++	+++	+++	+++

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Table 12.7: Grading of arterial pulse

<i>Finding</i>	<i>Grade</i>
Impalpable	0
Feeble	+
Palpable but diminished compared to the other side	++
Normal	+++
High volume or bounding pulse	++++

Additionally, the presence or absence of bruit and the palpability of abdominal aorta should be commented. The dorsalis pedis and posterior tibial arteries are impalpable in 2 per cent of normal people due to their anomalous course.

Bruit over an artery

In general the more severe the obstruction, the higher the frequency and length of the bruit (Table 12.8). Additionally the skull, the liver and any other site which was injured, operated or punctured should be auscultated for any evidence of arteriovenous fistula. In a child with unexplained congestive heart failure the skull should be auscultated for an intracranial AV fistula.

Table 12.8: Bruit over an artery

<i>Severity of obstruction</i>	<i>Nature of bruit</i>
Less obstruction	Short systolic bruit
More obstruction	Continuous bruit
	High pitched
	No bruit

Table 12.9: Sites of auscultation for arterial bruit

<i>Artery involved</i>	<i>Site of bruit</i>
Internal/external carotid	Anterior neck
Subclavian	Supraclavicular
Axillary	Infraclavicular
Thoracic aorta	Left interscapular
Abdominal aorta	Epigastrium
Celiac	Epigastrium
Renal	Upper abdominal quadrants, lumbar
Aorto-iliac bifurcation	Umbilicus
Common femoral	Inguinal
Superficial femoral	Femoral triangles, anteromedial thighs
Popliteal	Popliteal fossa

Table 12.10: Normal time delay for pulse wave propagation

Carotid	30 msec
Brachial	60 msec
Radial	80 msec
Femoral	75 msec

Brachiofemoral delay

The brachiofemoral delay rather than radiofemoral delay appears to be more sensitive in the diagnosis of coarctation as there is already a delay of 15 milliseconds from the brachial to femorals in normals. Any further delay is easily detected (Table 12.10).

Unless the delay is more than 20 msec it is not appreciable clinically. Blood pressure measurement is a more sensitive indicator of coarctation of aorta than brachiofemoral delay. A radio-radial or brachio-brachial delay is suggestive of vascular obstruction anywhere from the innominate artery on the right or subclavian artery on the left to the site of palpation.

In patients presenting with intermittent claudication but palpable peripheral pulses, evaluation is incomplete unless the patient is exercised and the pulses sought for by palpation, auscultation for a bruit, and also measurement of blood pressure.

Since atherosclerotic vascular disease tends to be generalized, it is a good clinical practice to examine peripheral pulse thoroughly in all patients with coronary artery disease.

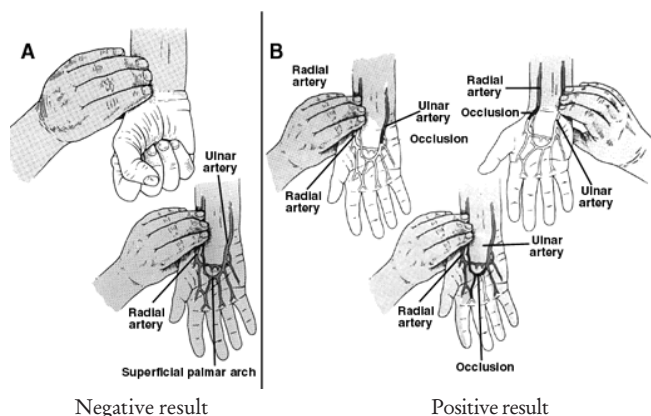
Allen test

Fig. 12.13: Allen test to demonstrate the presence of ulnar artery

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Evaluation of the patency of the radial and ulnar arteries supplying the hand is a pre-requisite for using the radial artery either for cardiac catheterization or as an arterial conduit for coronary bypass surgery. This test is intended to test the patency of the ulnar artery to maintain the circulation to the hand if the radial artery is occluded as a result of the procedure or harvested for a graft.

The result of the Allen test is considered normal when after compression of both ulnar and radial arteries, the hand colour returns to normal within 10 seconds after release of the radial artery.

THE ARTERIAL PULSE IN VARIOUS CLINICAL STATES

ARTERIAL PULSE IN A PATIENT WITH SHOCK

The pulse rate is usually rapid in shock. Disproportionate tachycardia should elicit the possibility of acute myocardial infarction, myocarditis, septic shock or an arrhythmia (Table 12.11).

The causes of an inappropriately rapid pulse rate could be:

- Acute myocardial infarction
- Myocarditis
- Septic shock
- Tachyarrhythmia

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A normal or diminished pulse rate in the setting of shock may suggest a

Table 12.11: The arterial pulse in shock

<i>Characteristic feature in pulse</i>	<i>Cause of shock</i>
Rapid, low volume	Cardiogenic shock Hypovolemic shock Septic shock
Paradoxical pulse	Pericardial tamponade Tension pneumothorax Acute large pleural effusion
Irregular pulse	Refer to atrial fibrillation
Pulse rate half of heart rate in ECG	'AFORMED'* phenomenon as in supraventricular tachycardias

*AFORMED = Alternate failure of response mechanical to electrical depolarization

vasovagal shock, complete heart block or drug induced bradycardia and hypotension. The causes of normal or diminished pulse rate could be:

- Complete heart block
- Drug induced
- Betablockers
- Calcium blockers
- Hypotension in patients with autonomic neuropathy
- Diabetes mellitus
- Idiopathic
- Shy-Drager's
- Under anesthesia
- Vasovagal
- Intracranial surgery

An irregular pulse in shock should suggest an arrhythmia like atrial fibrillation. An impression of irregularity is often created in patients with cardiac tamponade and paradoxical pulse but cardiac auscultation dispels the doubt. The causes could be:

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- Acute myocardial infarction
- Mitral stenosis
- Severe aortic stenosis
- Hypertrophic cardiomyopathy
- Acute pulmonary embolism
- Thyrotoxic crisis
- Acute cardiac tamponade
- Atrial fibrillation in WPW syndrome (rapid ventricular rate)

The paradoxical pulse of cardiac tamponade may be mistaken for atrial fibrillation.

Careful evaluation of the arterial pulse usually reveals the cause of shock in many situations. The characteristic features and causes are given in Table 12.11.

As can be seen, careful evaluation of the arterial pulse gives valuable clues regarding the etiology of shock.

ARTERIAL PULSE IN VALVULAR HEART DISEASE

The nature of arterial pulse in aortic stenosis and aortic regurgitation has already been discussed.

Arterial pulse in mitral stenosis

The arterial pulse in mitral stenosis is altered by the following variables.

- Rhythm
- Severity of mitral stenosis
- Associated
 - Aortic valve disease
 - Systemic hypertension
 - Anemia
- Age of the patient
- Presence or absence of systemic embolism

The irregularly irregular pulse of atrial fibrillation usually means severe mitral stenosis except in elderly patients, when AF can occur even with mild mitral stenosis. If the arterial pulse is regular in a patient with established atrial fibrillation on digitalis therapy, digitoxicity with AV nodal rhythm should be considered. Asymmetry or absence of peripheral pulses should particularly be checked for in all patients with mitral stenosis as evidence of systemic embolism. All patients with mitral stenosis and systemic embolism should have a transesophageal echocardiography to rule out left atrial thrombus as it is a contraindication to closed mitral commissurotomy or balloon dilatation. If peripheral embolism occurs with sinus rhythm, left atrial myxoma should be considered. In mild or moderate mitral stenosis, the pulse volume is normal. The typical low volume pulse occurs with severe mitral stenosis, severe PAH and right ventricular failure. Pulse volume can also be low with associated aortic stenosis or TS. High volume pulse in mitral stenosis occurs with associated aortic regurgitation, anemia or systemic hypertension. Rarely, a sharp carotid pulse may be simulated by severe tricuspid regurgitation with high RV pressures and peripherally transmitted venous pulse.

Arterial pulse in mitral regurgitation

The manifestations in the arterial pulse in mitral regurgitation depend on the following factors:

- Severity of mitral regurgitation
- Left ventricular function
- Etiology of mitral regurgitation
- Rhythm
- Associated aortic valve disease

Table 12.12: The arterial pulse in mitral regurgitation

<i>Characteristic pulse</i>	<i>Significance</i>
Normal volume collapsing pulse	Severe MR with good LV function
Bisferiens pulse	MR in association with HOCM
Attenuation of post-extrasystolic pulse (Brokenbrough sign)	
Slow rising pulse	AS mistaken for MR Functional MR with AS
Pulsus alternans	Secondary MR with cardiomyopathy or myocarditis
Irregularly irregular pulse of AF	Rheumatic MR
Slow but regular pulse	L-TGA with left AV valve regurgitation
Asymmetry of pulses	Infective endocarditis with systemic embolism

Severe mitral regurgitation with good left ventricular function results in normal volume collapsing pulse (Table 12.12). This is due to rapid ejection by the left ventricle with an advantage of lesser afterload and more preload.

With the onset of left ventricular dysfunction, the pulse loses its collapsing character. Atrial fibrillation in a young person with mitral regurgitation usually means rheumatic mitral regurgitation. Bisferiens pulse in mitral regurgitation should bring in the possibility of hypertrophic obstructive cardiomyopathy where mitral regurgitation is part and parcel of the lesion, or mitral regurgitation associated with aortic stenosis and aortic regurgitation.

Arterial pulse in congenital cyanotic heart disease

The arterial pulse may give a clue about the underlying lesion in this setting. Tetralogy or tetralogy-like lesions with right to left shunts, have normal or mildly increased pulse volume. Low volume pulses are a feature of severe defects like hypoplastic left heart syndrome or any of the conditions with heart failure. High volume or collapsing pulse occurs in some of these disorders (Table 12.13).

The ductus, bronchopulmonary collaterals and systemic to pulmonary artery shunt often are responsible for the aortic run off in most of these disorders. In tetralogy of Fallot, the left arm pulse is absent after left subclavian artery to pulmonary artery shunt.

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Table 12.13: Causes and mechanisms of collapsing pulse in cyanotic heart disease

<i>Condition</i>	<i>Mechanism(s)</i>
Truncus arteriosus	Truncal run off into pulmonary artery Truncal insufficiency
Pulmonary atresia	Bronchopulmonary collaterals
Tetralogy of Fallot	Bronchopulmonary collaterals Associated ductus After systemic to pulmonary artery shunt Associated aortic regurgitation

ARTERIAL PULSE IN CORONARY ARTERY DISEASE

Evaluation of the arterial pulse in coronary artery disease has more practical utility than in any other cardiovascular disease. The findings of the arterial pulse help in the differential diagnosis, diagnostic testing strategies, selection and dosage of drug therapy, access to invasive testing and interventions, as well as the surgical approach.

During acute coronary syndromes the pulse rate is usually increased but may decrease if the right coronary or the circumflex arteries are the culprits. The pulse rate is often used as a guide to select drugs in coronary artery disease. When the resting pulse rate is less than 60 per minute, betablockers are usually contraindicated and are never given if the pulse rate is less than 50/minute. Betablockers are the choice when the resting pulse rate is normal or higher. Amlodipin or nifedipine can be given safely in patients with a slow pulse rate.

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Arterial pulse in a patient with acute chest pain

In patients with chest pain as a presenting feature, the arterial pulse gives valuable information of use in the diagnosis and decision making (Table 12.14).

In view of the above mentioned reasons, the peripheral pulses in patients with CAD should be carefully evaluated.

In conclusion, examination of the arterial pulse is often the inaugural part of the physical examination. It is at this time the doctor touches the patient physically first time. The advantage of starting the examination from the dorsalis pedis pulses is that one can avoid missing palpating the peripheral pulses.

Table 12.14: The significance of the arterial pulse in a patient with chest pain

<i>Feature of arterial pulse</i>	<i>Significance</i>
Slow rising pulse	Severe fixed AS with angina
Bisferiens pulse	Hypertrophic cardiomyopathy AS with AR
Paradoxical pulse	Pericardial tamponade Acute tension pneumothorax
High volume or collapsing pulse	Severe AR of any cause (especially, syphilis) Anemia precipitating or aggravating angina Aortic dissection with AR
Asymmetry of pulses	Aortic dissection Acute MI with embolism Aortic valve disease with infective endocarditis
Femoral bruit/diminished pulses in lower limbs	Peripheral vascular disease Vascular access for catheterization/ angiography from upper limb Patient unsuitable for IABP or PCPS Associated claudication may mask angina Betablockers may aggravate claudication
Carotid bruit/diminished carotid pulse	Carotid evaluation along with coronary arteriography History of stroke or TIA increase the risk of stroke during CABGS Carotid surgery may have to be combined with CABGS
Diminished or absent left or right upper limb pulses	Left subclavian arterial obstruction is a contraindication to LIMA/RIMA grafting in CABGS Poor graft flow in LIMA/RIMA

PRACTICE IMPLICATIONS

- The most common error in evaluation of arterial pulse is missing to palpate the peripheral pulses.
- When the patient presents with intermittent claudication with palpable peripheral arterial pulse, peripheral arterial disease is not ruled out unless pulses are evaluated after exercising the patient. Peripheral pulse has to be examined by palpation, auscultation and measurement of blood pressure both at rest and after exertion.
- Improper recording of pulse rate is more common than is realized.
- Always look for pulsus paradoxus in all patients with hypotension or shock.

THE ARTERIAL PULSE

- If the left arm pulsus are impalpable, left internal mammary (LIMA) grafting may be contraindicated.
- Chronotropic incompetence is defined as a slow resting pulse that fails to accelerate normally with exercise. When it is not due to betablockade, it indicates sick sinus syndrome.
- Sinus node deceleration, defined as initial increase followed by subsequent decrease in heart rate during continued exercise, is a marker of right coronary artery disease.

13 Blood Pressure

Blood pressure is the product of cardiac output and peripheral vascular resistance and is a reflection of the performance of the heart and responsiveness of the vascular system. Elevation of arterial pressure as a physical sign is unique because the very elevation of it suggests the diagnosis of systemic hypertension, which is one of the most common cardiovascular disorder in adult population. Systemic hypertension is a major risk factor for coronary arterial disease, cerebrovascular disease and renovascular disease.

Blood pressure is expressed as mmHg or kPa
(1 mmHg = 0.13332 kPa; 1 kPa = 7.5006 mmHg).

The factors that determine arterial pressure are:

- Cardiac output
- Systemic vascular resistance
- Compliance of the vascular system
- Competence of the aortic valve
- Isolation of the arterial from venous system by the arterioles

Blood pressure in the arteries is the product of cardiac output and systemic vascular resistance.

Measurement of blood pressure

Among the various methods available for measuring blood pressure the auscultatory technique is the most commonly used. The other methods are:

Non-invasive methods (indirect)

Palpation

BLOOD PRESSURE

Auscultation
 Flush
 Ultrasound Doppler
 Oscillometric
Invasive method (direct)
 Intra-arterial

Non-invasive methods: Prior to any discussion on blood pressure recording it is essential to understand the nature of Korotkoff sounds and the requirements for the cuff used to measure blood pressure.

Korotkoff sounds are the sounds audible over the artery when a compressing cuff is gradually released. The vibrations produced by the flow of blood at different levels of release of cuff produce these sounds. There are five phases of Korotkoff sounds (Table 13.1).

The sphygmomanometer

The pneumatic cuff permits controlled occlusion of the underlying artery when inflated. A mercury or aneroid manometer is connected to a rubber bladder encased in a non-distensible outer cuff. The bladder length should be at least 75 per cent

Table 13.1: Korotkoff sounds

Phase	Description	Comment
Phase I	Onset of auditory sound Clear tapping sound	At peak systolic pressure
Phase II	Onset of swishing sound or murmur	Onset 10–15 mmHg lower from peak systolic pressure
Phase III	A louder murmur-like sound	Auscultatory silent gap occurs when phase II sounds are fainter or not heard 15–20 mmHg below phase II
Phase IV	Sudden muffling of sounds	Occurs 5–10 mmHg above the true diastolic pressure. Represents diastolic pressure in severe aortic regurgitation (AR) and hyperkinetic states
Phase V	Disappearance of sounds	Represents true diastolic pressure except in severe AR and hyperkinetic states

Table 13.2: Suggested sphygmomanometer cuff size in centimeters (modified from AHA)

<i>Patient subset</i>	<i>Arm circumference (mid-arm)(cm)</i>	<i>Bladder width (cm)</i>	<i>Bladder length (cm)</i>
Newborn		2.5–4.0	5–10
Infant		6–8	12–13.5
Child	13–20	9–10	17–19
Small adult	17–26	11–12	19–22
Standard adult	24–33	12–13	22–24
Large adult	33–42	15–17	32–33
Thigh	42–50	18–20	35–42

of the arm circumference. The cuff should be long enough to be wrapped around the arm (Table 13.2).

The cuff width should be at least 20 per cent larger than the arm diameter. The cuff for the thigh should be at least 15 cm in width and twice that in length.

Methods of measuring blood pressure

1. Palpation: The palpatory method allows a quick assessment of the systolic pressure though the systolic pressure estimated is about 10 mmHg lower than the intra-arterial or auscultatory method. Sometimes, in infants and young children this may be the only method possible. The diastolic pressure cannot be measured by this method. The systolic pressure estimated by this method is used as a guide to inflate the cuff above systolic pressure.

2. Auscultatory method: The cuff should be quickly inflated to 20 mmHg above peak systolic pressure estimated by palpatory method. The deflation should be slower. The deflation rate can be at the rate of 2–3 mmHg per second. The first appearance of Korotkoff sounds represents the systolic pressure and the point of disappearance is taken for diastolic pressure. In situations where the Korotkoff sounds are heard to the level of zero, the level of muffling should be taken as the diastolic pressure but a note should be made of this feature. For example, in a patient with severe AR, the BP should be expressed as 150/40–0 mmHg, indicating that muffling occurred at 40 mmHg but the sounds continue to be audible until zero. If more than one measurement is made, the cuff should be fully deflated and the arm emptied of venous blood before the next measurement. Venous pooling can muffle or drown out the Korotkoff sounds.

3. Doppler method: With the ultrasonic transducer placed over the artery, the

cuff pressure is deflated slowly as in the auscultatory method. An audible signal is appreciable over the artery when the cuff pressure is lower than the intravascular pressure. The sound signal disappears when the cuff pressure drops below the diastolic pressure. This method is very accurate for the estimation of systolic pressure but the diastolic pressure cannot be measured accurately.

4. Oscillometric method: This method permits estimation of systolic, diastolic and mean arterial pressures as well as the heart rates digitally. Recordings can be automatically repeated when required. Technical artifacts are common and the mean pressure is most accurate with this method. The systolic and diastolic pressures can be calculated from the mean pressure.

5. Flush technique: The principle of this method is similar to that of cuff application in the other methods. The limb is elevated, and an elastic bandage is applied from the finger tips or toes proximally to eliminate blood from the skin capillaries and veins to blanch the distal forearm and hand or lower leg and foot. With the bandage still applied, the BP cuff is inflated at least 20–30 mmHg above the expected systolic pressure. With the BP cuff inflated, the bandage is removed. The distal limb should now be blanched white. The cuff is deflated by 2–3 mmHg per second, and the pressure at which the first blush appears in the limb is taken as the blood pressure. The reading obtained is closer to mean arterial pressure than to peak systolic pressure. This method is most useful in infants with suspected coarctation of the aorta. In the presence of severe coarctation, this may be the only method possible, short of direct intravascular measurement.

Patient factors: The patient should be relaxed and shouldn't have taken medications which increase pressure. On the first visit, BP should be measured in all the four limbs in adults and at least in one upper limb and one lower limb in children. The other guidelines are listed in Table 13.3.

One of the methods of avoiding anxiety related rise in pressure is to apply the cuff at the beginning of physical examination, and measure the BP at the end of examination by which time the patient is accustomed to the idea of having a cuff tied around the arm.

Right arm and left arm pressures: Normally, the difference between the right and left upper limb pressures may be up to 10 mmHg. A significant pressure difference may occur in a variety of clinical situations and are listed in Table 13.4.

Table 13.3: Guidelines for measuring blood pressure

Patient factors	<p>Patient relaxed</p> <p>Quiet, comfortable room</p> <p>No coffee or smoking prior to measurement</p> <p>Avoidance of drugs which elevate pressure (nasal decongestants, cough syrups containing ephedrine, tricyclic antidepressants)</p> <p>Full and correct information of the dosage and nature of antihypertensive therapy</p>
Posture	
First visit	Supine + Standing
Revisit with drugs	Supine + Standing + Sitting
Follow up visit	Sitting
Any visit with postural symptoms	Supine + Sitting + Standing
Technical	<p>Appropriate cuff size encircling and covering two-thirds of the arm</p> <p>Mercury manometer is the choice</p> <p>Aneroid electronic equipment require calibration from time to time.</p> <p>For infants and children use appropriate sized cuff</p>
Which limb to use?	<p>All 4 limbs in the first visit in adults, one upper and one lower in children</p> <p>In the first visit take pressure in both arms; Followup: use the arm with the higher pressure</p>
When to take pressure?	<p>Don't take pressure the moment the patient enters the room</p> <p>Apply the cuff at the beginning of physical examination and take pressure at the end</p>
Attitude and manner	<p>Do not be in a hurry</p> <p>Make the patient feel as comfortable as possible</p>
How many readings?	<p>At least two recordings in first visit</p> <p>If the readings vary by more than 5 mmHg, take additional readings until two are close</p> <p>For a diagnosis of hypertension at least 3 sets of readings at least a week apart are needed.</p>
How to measure pressure?	<p>Inflate the cuff quickly to a pressure of 20 mmHg above systolic guided by radial pulse</p>

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Inaudible Korotkoff sounds	<p>The rate of deflation to be 2–3 mmHg/sec</p> <p>Disappearance of sounds (Phase V) is taken for diastolic pressure in adults</p> <p>Muffling (Phase IV) is taken as diastolic pressure in children</p> <p>Elevate the arm, open and close the fist for 4–6 times to empty the arm of pooled blood, and rapidly inflate the cuff</p>
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Table 13.4: Conditions under which there may be disparity in pressures between two arms

<i>Condition</i>	<i>Mechanism</i>
Normal variation	Orientation of origin of right arm and left arm arteries Right arm pressure is usually higher by 10mmHg
Arterial occlusion	Cardiogenic embolism Atherosclerosis Takayasu's arteritis Subclavian steal syndrome
Coarctation of aorta	Left subclavian arising from coarct segment Aberrant right subclavian artery distal to coarct segment
Dissecting aneurysm of aorta	Involvement of right innominate or left subclavian arteries
Thoracic outlet syndrome	Arterial compression
Supravalvular aortic stenosis	Jet of aortic ejection directed to right innominate artery
Normal fluctuations in pressure between two readings	

When measuring pressure most doctors have a tendency to use the figures that are most 'popular'. For example, 120/80 mmHg is the most popular normal pressure and is often the figure most often recorded. As the upper limit of normal is 140/90 mmHg, any reading slightly above this is often mentioned as lower than this to 'avoid' the diagnosis of hypertension.

Common errors

- Failure to measure blood pressure
- Failure to enter the correct reading in the case record even after measurement
- Inflating and deflating the cuff too rapidly

- Failure to concentrate on the mercury column while deflating
- Defective BP apparatus
- Reliance on electronic apparatus
- Cuff applied too loosely
- Inappropriate size of the cuff
- Failure to recognize 'auscultatory silent gap'
- Absence of Korotkoff sounds due to venous congestion
- Failure to keep the arm and BP apparatus at the level of the heart
- Measuring pressure as soon as the patient enters the room

The commonest error in measurement of blood pressure is surprisingly not measuring it at all. All patients who enter a hospital or a doctor's office should have their blood pressure measured irrespective of their symptoms. By not measuring blood pressure, the commonest cardiovascular disease in adults, systemic hypertension, is missed.

ALTERATIONS IN BLOOD PRESSURE

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SYSTEMIC HYPERTENSION

Systemic hypertension is a risk factor for coronary, cerebral and renovascular disease. Careful control of pressure reduces the vascular complications. The diagnosis of this common and potentially life-threatening disease is missed if blood pressure is not measured and classified accurately (Table 13.5).

Table 13.5: Classification of blood pressure for adults (18 years and older)

<i>Category</i>	<i>Systolic</i>	<i>Diastolic</i>
Normal	<130	<85
High normal	130–139	85–89
Hypertension		
Stage 1 (mild)	140–159	90–99
Stage 2 (moderate)	160–179	100–109
Stage 3 (severe)	180–209	110–119
Stage 4 (very severe)	>210	>120

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POSTURAL HYPOTENSION

In a normal person, the arterial pressure is maintained within physiological limits in all positions. With a change in position from supine to standing, there is a slight fall in systolic pressure not exceeding 5 mmHg, and the diastolic pressure remains constant or may rise slightly. These changes revert to normal levels within 3–5 minutes in normal individuals. This is made possible by the increase in systemic vascular resistance and heart rate. The integrity of the autonomic nervous system is essential to bring about these changes.

Postural hypotension is defined as a fall of 20 mmHg or more in systolic pressure, or 10 mmHg or more in diastolic pressure in upright posture. If the patient is symptomatic, even a marginal decrease should be considered significant.

Symptoms

- Light headedness
- Dizziness
- Blurring or loss of vision
- Extreme weakness
- Syncope

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Technique of measurement

- Supine BP and heart rate after the patient is supine for at least 5 minutes
- Standing BP immediately, and at 2–5 minutes

Table 13.6: Cardiovascular alterations in upright posture

<i>Alteration</i>	<i>Mechanisms</i>
Increased heart rate Sinus tachycardia	Diminished venous return Fall in stroke volume Fall in cardiac output Lesser distention of carotid stretch receptors Less reflex inhibition from carotid baroreceptor on the vasomotor centre in the brain stem
Blood pressure Systolic Diastolic	Fall of less than 5 mmHg Constant or rise of less than 5 mmHg

- Continue to measure BP upto 10 minutes if postural hypotension is suspected

The clinical diagnosis should take into consideration not only the blood pressure changes but also the symptoms. The commonest cause of postural hypotension in clinical practice is that induced by antihypertensive medication. Hypovolemia remains another common cause. An intact autonomic nervous system is essential for maintenance of arterial pressure in general, and particularly in an upright position.

Causes

A. Drugs

Antihypertensive

Centrally acting: Methyl dopa, clonidine

Adrenergic blockers: Guanethidine

Alpha blockers: Phenoxybenzamine, labetalol

Vasodilators: Prazosin, hydralazine

ACE inhibitors

Diuretics

Calcium channel blockers: Nifedipine

Antianginal agents: Nitrates/nitroglycerine

Antidepressants: Tricyclic antidepressants, monoamine oxidase inhibitors

Tranquilizers/Sedatives: Barbiturates, phenothiazines

Selective neurotoxic drugs: Alcohol

B. Hypovolemia

Diuretics

Dehydration due to any cause

Hemorrhage

Excessive perspiration

Overdialysis

Idiopathic hypovolemia

C. Endocrine disorders

Addison's disease

Hypoaldosteronism

Pheochromocytoma

Renovascular hypertension

D. Vascular insufficiency

Varicose veins

Absent venous valves

Arteriovenous malformations

E. Vasodilator excess

Mastocytosis (histamine, prostaglandin D₂)

Hyperbradykininism (bradykinin)

Carcinoid (bradykinin)

Hypermagnesemia

F. Autonomic neuropathy

Primary

Primary autonomic failure (Bradbury Eggleston syndrome)

Shy–Drager syndrome

Autonomic failure with Parkinson’s disease

Riley–Day syndrome (familial dysautonomia)

Baroreflex failure

Acute pandysautonomia

Secondary autonomic neuropathies

Diabetes, alcoholism, amyloid

Autoimmune disorders

Guillain–Barre syndrome

Acute/subacute dysautonomia

Mixed connective tissue disease

Rheumatoid arthritis

Eaton–Lambert syndrome

Systemic lupus erythematosus

Carcinomatous autonomic neuropathy

Porphyria, Fabry’s disease, Tangier’s disease

B12 deficiency

Hereditary sensory neuropathies

CNS infections

Syphilis, Chagas' disease, herpes zoster, HIV infection, botulism

Spinal cord lesions

Familial hyperbradykininism

Cerebral/mid-brain lesions: Vascular lesions or tumours involving hypothalamus and mid-brain (craniopharyngioma), multiple sclerosis, Wernicke's encephalopathy, Adie's syndrome

Spinal cord lesions

Advanced age

Dopamine hydroxylase deficiency

Renal failure

Elderly patients are particularly susceptible to volume depletion and adverse effects of drugs because of associated cerebrovascular disease, decreased baroreceptor sensitivity, decreased muscle tone and increased renal sodium loss. The diagnosis of postural hypotension is missed if blood pressure is not measured in *standing position*. One must look for this sign in certain clinical situations:

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- All patients on antihypertensive medication on follow up
- All patients on vasodilator therapy for heart failure
- All patients with syncope or near syncope
- Patients presenting with chronic fatigue or weakness
- Patients diagnosed to have depression
- Hypovolemia due to any cause
- Hemorrhage either external or internal

Postural hypotension is an early sign of hypovolemia due to any cause before frank hypotension supervenes.

HYPOTENSION AND SHOCK

When the systolic blood pressure is less than 90 mmHg, hypotension is said to exist. Shock is a clinical syndrome with hypotension as an important but not essential part of the clinical picture. The traditional definition of shock as equivalent to low arterial pressure, and excessive focus on arterial pressure, results in many cases in misdiagnosis and mismanagement of patients with shock.

Shock is a syndrome, a constellation of symptoms and signs that are subjective

and imprecise. A low arterial pressure is only one of the signs of shock. Hypotension, though common, is not essential for a diagnosis of shock.

Signs and symptoms

- Hypotension or normotension
- Pallor
- Cold, clammy skin
- Tachycardia
- Altered mental status
- Oliguria

Though hypotension is common, normal blood pressure or even hypertension can occur in the initial stages of shock. Fall in pressure can be precipitous in such situations. In the presence of shock, or low perfusion states, indirect measurement of pressure by the cuff method is imprecise and can be misleading. With extreme vasoconstriction of shock, the central aortic pressure is often higher (by as much as 40 mmHg) than peripheral arterial pressure.

Routine cuff method is unreliable and can be misleading in this setting. Though direct invasive pressure monitoring is superior to indirect methods (Table 13.7), errors can occur even with the direct method due to improper techniques or instrumentation.

Hypotension is often a danger signal suggesting a serious inadequacy of the circulatory system. Prompt recognition and management is the key to successful

Table 13.7: Unreliability of indirect pressure recording in shock

<i>Cause</i>	<i>Mechanism</i>
Adrenergic vasoconstriction	Gradient of pressure between central aorta and peripheral arteries (upto 40 mmHg)
Reduction in stroke volume, increased vascular resistance	Diminished or absent Korotkoff sounds

Indications for direct intra-arterial pressure monitoring

- Shock
- Nitroprusside infusion
- Hypertensive crises
- Acute mitral regurgitation

Indications for measuring blood pressure immediately in hospitalized patients

- Restlessness
- Drowsiness
- Excessive sweating
- Weakness
- Unexplained tachycardia or bradycardia
- Respiratory distress
- Elderly patients exposed to any hypotensive medication
- Identification of high-risk patients and monitoring them frequently

outcome in this setting. Though blood pressure is measured routinely in all patients coming to the outpatient or emergency room, this is not often the case with patients who are already hospitalized. For a variety of reasons, onset of hypotension is not promptly recognized in hospitalized patients. The box above summarizes the situations where blood pressure should be measured immediately in hospitalized patients.

The common story is that the nurse when called to see a restless patient in the night gives a sedative (diazepam) without informing the doctor. The doctor in turn when called does the same only to realize several hours later that the beginning of hypotension was missed.

The moment hypotension is detected, one should look for causative factors. The clues to the diagnosis often come from the history and the rest of the physical examination. The importance of clinical examination is often not realized in patients with shock.

Clinical evaluation

In patients with trauma and loss of blood, the clinical signs may help estimate the volume of blood lost (Table 13.8).

State of consciousness	Restlessness, anxiety,
Skin	Temperature, moistness, color and turgor, rash
Mucous membranes	Colour and moistness
Nail beds	Colour and capillary filling
Peripheral veins	Collapsed or distended
Jugular venous pulse	Collapsed or distended
Pulse	Rate and rhythm
Respiration	Rate and depth
Urine	Hourly output

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Table 13.8: Clinical correlates in hemorrhagic shock

<i>Vital sign</i>	<i><15%*</i>	<i>15–30%*</i>	<i>30–40%*</i>	<i>>40%*</i>
Heart rate	<100	>100	>120	>140
Systolic BP	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal/ increased	Decreased	Decreased	Decreased/ absent
Capillary refill	Normal	Delayed	Delayed/ absent	Absent
Respiratory rate	14–20	20–30	30–40	40–50
CNS, mental state	Anxious	More anxious	Anxious/ confused	Confused/ lethargic

* Blood loss as volume percentage

Table 13.9: Causes of shock and clues to the diagnosis

<i>Cause</i>	<i>Clues to diagnosis</i>
Hypovolemic shock Diarrhea, vomiting, hemorrhage, Diuretics	Sinus tachycardia Cold extremities No elevation of jugular venous pressure No ventricular gallop
Cardiogenic shock Myocardial infarction, acute myocarditis	Sinus tachycardia Cold extremities Elevated jugular venous pressure Ventricular gallop Crepitations at lung bases
Arrhythmia	Heart rates too high or too low or impalpable Abnormal wave pattern in jugular venous pressure
Obstructive shock Acute pulmonary embolism	Dyspnea is the rule Sinus tachycardia Cold extremities Elevated jugular venous pressure Dry lungs Right ventricular S3/S4
Compressive shock Pericardial tamponade Tension pneumothorax Bilateral large pleural effusions	History of infection or trauma Sinus tachycardia Tachypnoea Cold extremities

<i>Cause</i>	<i>Clues to diagnosis</i>
Distributive shock Septic shock Anaphylactic shock Drugs, radiologic contrast Bee sting, Poison ivy Drug induced Antihypertensive agents Calcium blockers Betablockers Nitrates Phenothiazines Barbiturates (suicidal intent) Endocrine shock Addison's disease Panhypopituitarism Pheochromocytoma (adrenaline secreting) Vasovagal shock	Elevated jugular venous pressure Pulsus paradoxus No gallop Dry lungs Sinus tachycardia Warm extremities Fever may be present Evidence of infection History of drug allergy/bronchial asthma/skin allergy Evidence of exposure to a drug/agent Bronchospasm Skin rash/itch/angioedema Sinus tachycardia Warm extremities Sinus tachycardia Bradycardia with betablockers or calcium blockers Warm extremities Other systemic effects of drug Long term steroid use/acute stress pallor Paroxysmal hypotension with signs of adrenergic excess Sinus bradycardia Hypotension

In the majority of patients, the diagnostic clues as to the cause of shock are often available from the clinical examination (Table 13.9).

As the outcome is often dependent on the measures taken in the initial few minutes or hours, a practical 'first things first' approach is useful. As anaphylactic shock is rapidly fatal and responds to adrenaline and steroids promptly if given in time, never waste time on detailed evaluations if there is any element of doubt. Apply the same rule to the adrenal crisis, which responds dramatically to intravenous steroid.

BLOOD PRESSURE

Table 13.10: Initial approach to a patient with shock

<i>Step</i>	<i>Approach</i>
Rule out conditions simulating shock	Confirm the presence of shock Rule out peripheral vascular disease with diminished pulses
Treat conditions which are rapidly fatal but respond dramatically to specific therapy	Rule out vasovagal shock (if in doubt give atropine and volume infusion) Rule out anaphylactic shock (If in doubt, give adrenaline and steroid IV) Rule out addisonian crisis (If in doubt give steroid IV) Rule out cardiac tamponade (If in doubt, insert a needle into the pericardium) Rule out tension pneumothorax (If in doubt, insert a needle into the pleural cavity) Always consider septic shock in the differential diagnosis even in afebrile patient
Take care of volume	Infuse fluid if in doubt, as hypovolemia is a common cause of shock, even in cardiovascular disorders
Take care of ventilation	Ensure adequate ventilation

This combined diagnostic and therapeutic approach is ideally suited to a condition like shock because, any delay can cause irreversible damage to vital organs.

Clinical recognition and management of anaphylactic shock

- Sudden unexpected tachycardia (increase of > 20 beats/minute) or hypotension > 30 mmHg fall in pressure, especially if a drug has been given within the preceding 10 minutes.
- Skin rash, angioedema and bronchospasm are confirmatory
- Stop further administration of the drug
- Airway maintenance with 100% oxygen, if the patient is not ventilated, use mask ventilation
- The first drug of choice is epinephrine 2.5 ml 1:10,000 dilution every 5–10 minutes
- Start maintenance infusion 1–20 µg/kg body weight
- Inject 200 mg of hydrocortisone intravenously
- Volume expansion: rapidly infuse 2–10 litres of crystalloid. Appropriate venous access may be necessary
- Intravenous antihistamine (H1 and H2) may be given if the reaction persists
- Give bicarbonate early based on acid-base balance

Some of these criteria are particularly helpful in recognizing the disorder during anesthesia, cardiac catheterization, postoperative state, cerebrovascular accidents or in patients under sedation. To confirm the diagnosis, plasma histamine levels are helpful. A sample of 2–5 cc blood is collected in an EDTA tube and kept on ice immediately. The plasma is then separated and stored at -70°C , to be assayed later. The plasma tryptase is an enzyme released during mast cell degranulation. The levels of this enzyme persist for many hours and can be estimated serially over a 24 hour period. In initial studies of 30 severe reactions, levels of 1–2 ng/ml tryptase were associated with significant hypotension.

In the cardiac catheterization laboratory, the best way to treat these patients is to be aware that these reactions are possible due to iodine containing contrast agents. The high-risk patient can be identified if he has a history of allergies. Betablocker therapy appears to increase the tendency for anaphylaxis. Pre-treatment with steroids diminishes the risk. All catheterization laboratories should keep pre-diluted and preloaded injections of adrenaline each morning before the procedures are started (to save time should the need arise). Routine femoral venous sheath is particularly valuable as a reliable access in case of shock to administer large volume of fluid.

PULSUS PARADOXUS

Pulsus paradoxus is best elicited with the sphygmomanometer. It is an exaggeration of normal fall in systolic pressure in inspiration. The patient is asked to breath normally. The cuff is inflated to above the level of systolic pressure and is deflated very slowly (2–3 mmHg/sec). The point at which the Korotkoff sounds are intermittently heard is noted. By further deflation, the sounds are continuously audible during both inspiration and expiration. The difference between the two points is measured as the degree of paradoxus. In normal people the inspiratory fall in pressure is not more than 8 mmHg. Any exaggeration above this value is considered a positive sign.

Causes

Normal

- Pregnancy
- Exaggerated voluntary inspiration (student paradoxus)
- Extreme obesity

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Severe obstruction to airways

- Acute severe bronchial asthma
- Severe emphysema
- Upper airway obstruction

Pericardial disease

- Cardiac tamponade
 - Effusive constrictive pericarditis
 - Severe constrictive pericarditis
- Shock
- Hypovolemia

Sphygmomanometric paradoxus is not only a more sensitive sign than pulsus paradoxus, but also permits quantification of the degree of paradoxus in mmHg. This sign should be looked for in all clinical syndromes with any of the presenting features given below:

- Whenever palpable paradoxus is suspected
- In all patients with pericardial effusion
- Constrictive pericarditis
- Unexplained heart failure
- 'Heart failure' without S3
- Acute episode of asthma
- All patients with shock
- 'Unexplained' liver enlargement/Right hypochondriac pain
- All patients with syncope
- All patients with history of trauma (hemopericardium)

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When the Korotkoff sounds are feeble, the clue to the beginning of sounds or systolic pressure is oscillation of the mercury column with each pulse.

Blood pressure alternans (pulsus alternans)

This is characterized by a regular sinus rhythm with alternate strong and weak beats. It is often a sign of myocardial dysfunction and is related to alternating more and fewer contractile elements participating in each contraction. Alternans is most commonly seen in severe aortic stenosis with left ventricular dysfunction, but also occurs with any condition with severe ventricular dysfunction. Alternans can be total, involving both sides of the heart, or partial, involving only the right

or left ventricle. In aortic stenosis, the strong and weak beats are appreciated as variation in the intensity of systolic murmur alternatively. When the weak beat is too weak to be palpable, only auscultatory alternans is appreciable. Aortic regurgitation, systemic hypertension and reducing the venous return by head tilting or nitroglycerine usually exaggerate pulsus alternans and assist in its detection. Pulsus alternans is easier to detect by sphygmomanometer than by palpation of the arterial pulse. Palpable alternans over the peripheral pulse is detectable only when the aortic pressure alternates by more than 20 mmHg. The Korotkoff sounds alternate in their intensity with alternating beats. Electrical alternans is an independent phenomenon and has no relationship to pulsus alternans.

Causes of blood pressure alternans

- Severe aortic stenosis
- Dilated cardiomyopathy
- Myocarditis
- Severe AR with left ventricular failure especially after aortic valve replacement
- Any cause for ventricular dysfunction

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The purpose of describing this physical sign in the chapter on blood pressure is that alternans of blood pressure is a more sensitive sign than palpable pulsus alternans. *One must look for this sign in all patients with aortic stenosis and heart failure while measuring blood pressure.*

BLOOD PRESSURE IN AORTIC REGURGITATION

In aortic regurgitation, the diastolic pressure falls and the systolic pressure increases as the severity of regurgitation increases. As a result, the pulse pressure increases. When the pulse pressure is above 70 mmHg, severe aortic regurgitation is likely. Diastolic pressure above 70 mmHg is unlikely in moderate to severe aortic regurgitation. The systolic pressure is often normal in children with severe aortic regurgitation but is commonly elevated in older patients with aortic regurgitation. Though the systolic pressure does not correlate well with the degree of aortic regurgitation in elderly patients, in younger patients elevated systolic pressure is often indicative of significant aortic regurgitation. Significant systolic hypertension in aortic regurgitation is a feature in elderly patients with rigid arterial system, associated systemic hypertension or coarctation of aorta.

The systolic blood pressure in AR correlates with left ventricular end-diastolic volume. The higher the systolic blood pressure, the larger the end-diastolic volume. In a recent study by Pirwitz et al, the higher PSP/ESV ratio is found to be a reliable predictor of symptomatic improvement after valve replacement.

If the systolic pressure is above 170 mmHg, in a patient with aortic regurgitation, associated aortic stenosis of significance is unlikely. Similarly, if the diastolic pressure in aortic regurgitation is 40 mmHg or less, severe free aortic regurgitation is likely and usually rules out associated AS of any significance. Accurate measurement of blood pressure in severe aortic regurgitation may be difficult due to the fact that the pistol shot sounds may be audible over the brachial arteries and gives an impression of very high systolic pressures nearing 300 mmHg. The Korotkoff sounds may be audible until zero giving an impression of zero level of diastolic pressure. However, the diastolic pressure is never zero in any degree of aortic regurgitation. The onset of Korotkoff sounds at systolic pressure is recognized by their sharper quality in comparison to the pistol shot sounds. Though the Korotkoff sounds are audible to the level of zero, the onset of muffling occurs around 40 mmHg and should be taken as diastolic pressure. The BP in severe aortic regurgitation is typically expressed as 130/40–0 mmHg, and implies that though the sounds are audible up to 0 mmHg, muffling occurred at 40 mmHg. By the mechanisms described above, the systolic pressure is overestimated and the diastolic pressure is underestimated in severe aortic regurgitation. Normalization of diastolic pressure with fall in pulse pressure can occur in aortic regurgitation with the onset of left ventricular failure.

Normally, the lower limb systolic pressure is 10–20 mmHg higher than the upper limb pressure. In aortic regurgitation and other conditions with aortic runoff, the lower limb pressure increases to above 20 mmHg. This increase in systolic pressure in the lower limb above 20 mmHg is called Hill's sign. The more the severity of aortic regurgitation, the more the systolic pressure in the lower limbs. The degree of abnormality or positivity of Hill's sign correlates well with the angiographic severity of aortic regurgitation.

Brachio-femoral or brachio-brachial delay is a less sensitive sign than blood pressure measurement in the limbs. The blood pressure should be measured in all the limbs in all the patients with aortic regurgitation because Hill's sign permits estimation of severity but any asymmetry of pressure gives a clue to the underlying coarctation of aorta or aortic dissection. An equal systolic pressure in upper and

Table 13.11: Angiographic correlations of Hill's sign in aortic regurgitation (AR)

<i>Lower limb systolic pressure</i>	<i>Evaluation of AR</i>
< 20 mmHg	Normal or Trivial or mild AR Angiographic 1+ AR Associated coarctation
20–40 mmHg	Angiographic 2+ AR
40–60 mmHg	Angiographic 3+ AR
> 60 mmHg	Angiographic 4+ AR

Table 13.12: Situations in which sphygmomanometer cuff is used as a venous tourniquet

<i>Indication</i>	<i>Method and mechanism</i>
Acute pulmonary edema	Cuff inflated around three limbs Reduction in venous return Inflation pressure to less than diastolic or 70–80 mmHg Rotate the cuff between the limbs
To bring out the murmur of AR	Cuff inflated around both lower limbs Increase in systemic vascular resistance

lower limbs may still suggest coarctation of aorta especially in the presence of significant aortic regurgitation.

BLOOD PRESSURE IN ACUTE MYOCARDIAL INFARCTION

A systolic blood pressure of less than 100 mmHg, at admission carries an unfavourable prognosis in acute MI. Uncontrolled severe hypertension is a contraindication to thrombolytic therapy in acute MI. Acute myocardial infarction with systemic hypertension is associated with low hospital mortality even without thrombolytic therapy.

OTHER USES OF THE SPHYGMOMANOMETER

Venous tourniquet

The sphygmomanometer cuff can be used to reduce venous return in certain conditions. The cuff is tied around both thighs and one of the arms and is inflated

BLOOD PRESSURE

to just below the level of diastolic pressure or 80 mmHg. The cuffs are kept inflated for about 15 minutes at a time and rotated through the other arm. This method is of value in treating acute pulmonary edema by reducing the venous return (Table 13.12).

Diagnosis of thrombocytopenia (Hess sign)

This test is intended to assess the capillary fragility. The cuff is tied around the arm and is inflated to a pressure midway between the systolic and diastolic pressures for 5 minutes. The number of petechiae appearing in an area of 2.5 cm² on the inner aspect of the arm, are counted. More than twenty petechiae are considered abnormal.

PRACTICE IMPLICATIONS

- Blood pressure should be measured in all patients who visit a doctor.
- Systemic hypertension is the commonest chronic cardiovascular disorder and can be missed if the BP is not measured.
- ‘Sphygmomanometric paradoxus’ is a more sensitive sign than pulsus paradoxus. Make it a habit to look for cardiac tamponade while measuring blood pressure.
- A normal blood pressure or even hypertension does not rule out shock syndrome or pericardial tamponade.
- Improper recording of blood pressure is more common than realized.
- Pressure is the force per area unit. Expressed as mmHg or kPa (kilo Pascal). KPa is not commonly used, except by some examiners.
- kPa = 7.5006 mmHg; 1 mmHg = 0.13332 kPa.
- A normal blood pressure or even severe hypertension does not rule out cardiac tamponade. Cardiac tamponade manifests in a wide clinical spectrum.
- Even when the blood pressure has been recorded earlier by somebody else, always re-record the blood pressure as a routine. Unreliable recording of blood pressure is more common than is realized. It happens at all levels of training.

When you measured the blood pressure and found it elevated, you made a very important cardiovascular diagnosis.

14 The Jugular Venous Pulse

The normal arterial pulse reflects the performance of the left ventricle and is easily available for evaluation. The filling pattern of the left ventricle is unavailable, as we do not have access to the intrathoracic pulmonary veins. The performance of the right ventricle is not available externally by pulmonary arterial pulse, but the filling pattern is readily available through the jugular veins. The student, as well as the physician, is often confused as to the method of measuring the level and recognizing the wave pattern in the jugular venous pulse (JVP). In this chapter, we intend to clarify certain concepts basic to the understanding of jugular venous pulse (Tables 14.1; 14.2).

Table 14.1: Information derived from the jugular venous pulse

<i>Information</i>	<i>Clue in jugular venous pulse</i>
Level of right atrial pressure Right ventricular filling pressure (RVF) Central venous pressure	Level of jugular venous pressure
Obstruction to superior vena cava	Elevated jugular venous pressure with absent pulsations
Pattern of RV filling or RA emptying	y descent
Rapid filling (RVF, TR, CP)	Rapid y descent
Slow filling (TS)	Slow y descent
Little or no filling (tamponade)	Obliterated y descent
Resistance to pre-systolic RA emptying (TS, non-compliant RV)	Prominent a wave
Hyperkinetic circulatory state (Anemia, thyrotoxicosis)	Venous hum
Sequence of atrioventricular contraction (AV dissociation, CHB, junctional rhythm)	Analysis of rhythm

(RVF: right ventricular failure, TR: tricuspid regurgitation, CP: constrictive pericarditis, TS: tricuspid stenosis, AV: atrioventricular, CHB: complete heart block)

THE JUGULAR VENOUS PULSE

Table 14.2: Relation of atrial pressure to waves in JVP

<i>Waves in JVP</i>	<i>Atrial pressure</i>	<i>Mechanism</i>
<i>a</i> wave	••	Atrial contraction
\times descent	•	Atrial relaxation; descent of AV
septum		
<i>v</i> wave	•	Atrial filling
<i>y</i> descent	•	RV filling/atrial emptying

The normal jugular venous pulse (Figs. 14.1; 14.2) consists of an *a* wave, \times descent, *c* wave, *v* wave and the *y* descent.

Normal jugular venous pattern

The positive wave just preceding the carotid upstroke and corresponding to the P wave on ECG results from atrial systole. This is an *a* wave. A small positive deflection may be recorded and is due to tensing of the tricuspid valve at the onset of systole. Sometimes it is considered to be due to the transmitted carotid pulse. The \times descent is due to the descent of the tricuspid valve during ventricular emptying in systole. The subsequent positive wave, the *v* wave, is due to the venous return to the atrium. With the opening of the tricuspid valve in early diastole there is sudden flow of blood from atrium into the ventricle resulting in the *y* descent. Any obstruction in superior vena cava will obliterate these wave forms.

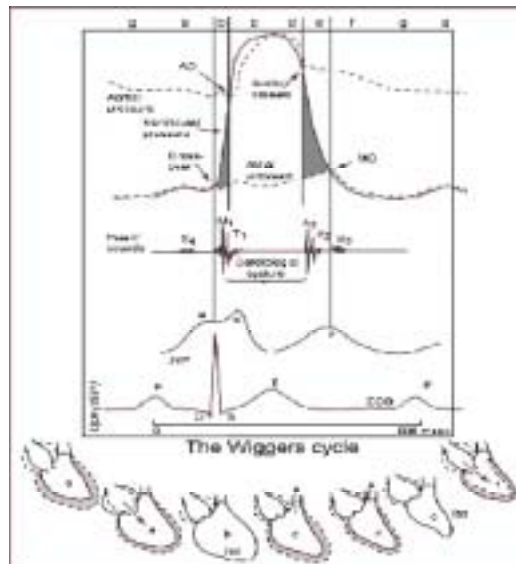


Fig. 14.1: Time relationship of wave pattern of JVP with other cardiac events

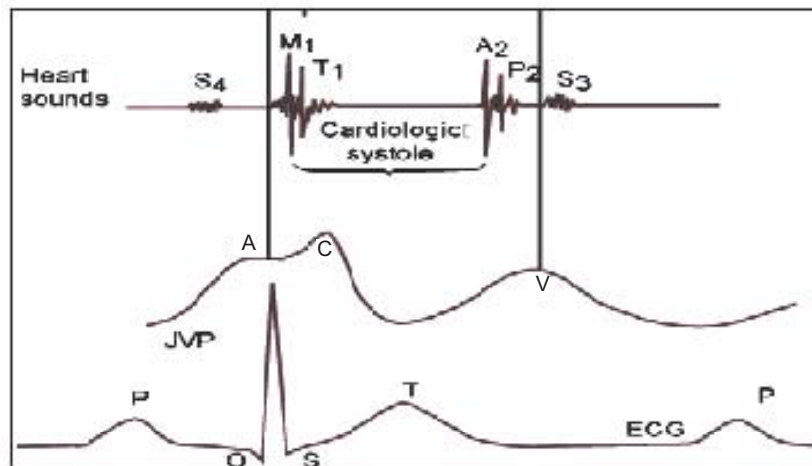


Fig. 14.2: Normal jugular venous pattern

The *c* wave is not appreciated by the bedside easily and is related to the transmitted carotid pulse when recorded in the neck, and due to tricuspid valve closure when recorded in the right atrium.

Table 14.3: Differentiating arterial and venous pulsations in the neck

Feature	Venous pulsations	Arterial pulsations
Site	Laterally located, superficial and widespread	More medial, deeper and localized
Changes with respiration, position and increased abdominal pressure	Alters with all these maneuvers	No alterations
Number of pulsations	Multiple	Single pulsations
Level	Definite upper level is visible	No definite upper level
Visibility and palpability	Better visible than palpable and is easy to obliterate (though in severe TR with high RV pressure, considerable pressure is needed to obliterate the <i>v</i> wave needed)	Better palpable than visible, difficult to obliterate
Ascents and descents	The descents are equally impressive or more impressive	The ascent or upstroke is impressive, the descent or downstroke is unimpressive
Associated features	PAH, PS, TR, RVH, RV failure	AR, hyperkinetic states

THE JUGULAR VENOUS PULSE

As a first step, one must differentiate arterial pulsations from venous pulsations in the neck (Table 14.3).

In general, if the pulsations in the neck are prominent and none of the peripheral arterial pulses are impressive, venous pulsations are likely.

EVALUATION

The following features have to be noted in the jugular venous pulse for optimal clinical information:

1. Level
2. Wave pattern
3. Respiratory variation in level and wave pattern
4. Hepatojugular reflux
5. Venous hum
6. Liver size and pulsations.

Level

Any measurement requires a point to start with and a point to end at. The starting point or the venous pressure is the centre of the right atrium. This is called the phlebostatic axis. The upper level of the venous column in the neck is the end point of venous pressure. By its intrathoracic location, the centre of the right atrium is not available for any direct measurement.

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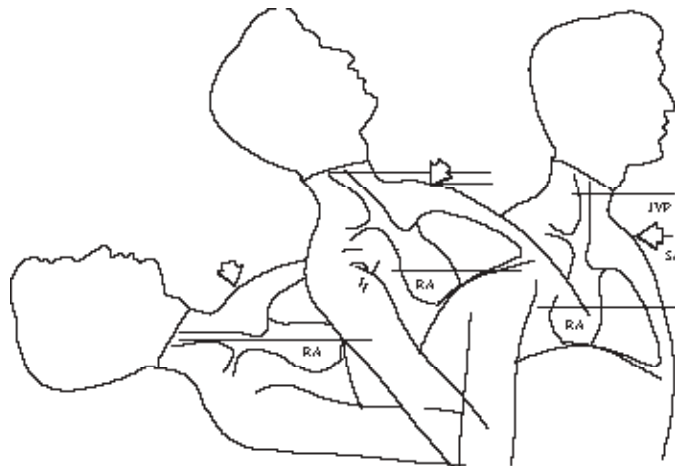


Fig. 14.3: Technique of measurement of jugular venous pressure
(Height of JVP=Height of venous column of sternal angle (SA) + 5 cm)

In an adult with anatomically normal chest, the sternal angle or angle of Louis is found to be 5 cm above the centre of the right atrium.

Technique of measurement of jugular venous pressure (Fig. 14.3): The jugular venous pressure (JVP) corresponds to the vertical height of the venous column from the centre of right atrium. Since the angle of Louis has a fixed relationship with the centre of right atrium, this landmark is used for measurement. The vertical height of the venous column can be measured either in the sitting position, if it is elevated, or in a semi-reclining position. In supine position, the upper level of the venous column is usually not seen, but the wave forms are best seen in this position.

As the sternal angle is readily available, all measurements of jugular venous pulse can be made starting from that point; adding 5 cm gives the total right atrial pressure. The sternal angle is available in all positions of the body. However, the upper level of the venous column is not available either in sitting or supine positions. In normal persons, in sitting position it is below the clavicle, and therefore cannot be seen. In supine position, the whole column moves beyond the angle of the jaw into the intracranial cavity and cannot be reached. (Since the whole column becomes horizontal, vertical height cannot be measured). To bring up the venous column from below the clavicle in the sitting patient, the patient is positioned at an approximate angle of 45 degrees on the bed. The angle should be subtended between the trunk and the bed, not at the neck. The neck and trunk should be in the same line. There is nothing sacrosanct about this angle as any angulation which permits viewing of the upper level of the venous column is appropriate. Two horizontal lines are drawn, one from the sternal angle and the other from the upper level of the venous column (in relation to the floor), and the vertical measurement between these lines is noted. If the venous column is seen above the upper border of the clavicle in sitting or standing position, direct vertical measurement from the sternal angle can be made and no horizontal line need be drawn. The jugular venous pressure is expressed as the number of cm above the sternal angle. The total pressure in the right atrium is obtained by adding 5 cm to it. Normal measurement does not exceed 4 cm above the sternal angle, hence it is hardly seen above the clavicle. By way of conversion, 1.3 cm column of water or blood is equal to 1 mmHg. Normal right atrial mean pressure does not exceed 7 mmHg (or 9 cm of jugular venous pressure by blood).

Normally the height of *a* and *v* waves is clearly seen and measured. Usual bedside assessment of JVP does not clearly indicate the mean pressure of the right atrium and only an approximation can be made.

The easiest way to detect an elevated JVP is to ask the patient to sit or stand, and if the venous column cannot be seen above the upper border of the clavicle, the JVP is not elevated. The internal jugular vein, between the two heads of the sternomastoid, should be used for measurements and the external jugular veins should be used only when they are pulsatile. The jugular venous pressure may be elevated when there is elevation of right ventricular end diastolic pressure, tricuspid stenosis, superior vena cava obstruction, compressive pericardial disorders or circulatory overload as in renal failure.

Causes of elevated JVP: The causes of elevated jugular venous pressure are:

- Superior vena cava obstruction
- Right ventricular failure due to any cause
- Tricuspid stenosis/regurgitation
- Pericardial compression (constriction/tamponade)
- Circulatory overload
- Renal failure
- Excessive fluid administration
- Atrial septal defect with mitral valve disease

Except in the setting of superior vena cava obstruction, all conditions producing elevated jugular venous pressure give rise to pulsations in neck veins. Rarely, in situations where jugular venous pressure is markedly elevated without associated tricuspid regurgitation, pulsations may be absent. Though superior vena cava obstruction is the most common cause of pulseless elevation of jugular venous pressure, cardiac tamponade and severe constrictive pericarditis may occasionally present in the same way. The causes of elevated JVP with little or no pulsations are:

No pulsations

- When external jugular veins are used
- Superior vena cava obstruction

Little or no pulsations

- Acute severe cardiac tamponade
- Severe constrictive pericarditis

In the clinical settings described above, there may be two problems:

1. The venous pulsations may be difficult to discern,
2. The elevation of jugular venous pressure may be missed for the same reason.

Wave pattern

The normal jugular venous pulse consists of two positive waves or ascents and two negative waves or descents. The best way to identify them would be to simultaneously auscultate and observe the wave pattern. Position the patient in such a way that the wave pattern is clearly seen. If the JVP is elevated, check pulse in the sitting position. If there is no rise in JVP, inclination of about 45 degrees brings out the venous column and wave pattern. Though the *a* wave in the jugular venous pulse occurs before the first heart sound, in actual appreciation by the bedside, the *a* wave appears to occur along with S1. This is due to the time delay between atrial contraction and transmission of the wave into the neck and also the intrinsic delays with left and right sided events. In fact, if the *a* wave appears to occur well before S1, the P-R interval is prolonged as in first degree heart block. The descent that follows S1 is α descent and *v* wave coincides with S2. The *y* descent follows the S2. In individual patients either the ascents or the descents are impressive. One can use either the wave or the descent to identify the pattern. As an example if the *y* descent is detectable the wave that precedes this must be the *v* wave and the wave that precedes the α descent must be the *a* wave. In the normal jugular venous pulse, the *a* wave (due to active atrial contraction) is sharper and more prominent than the *v* wave (due to passive atrial filling). One must practice looking at the jugular venous pulses of as many normal people as possible to attain proficiency. The *y* descent is more prominent than the α descent in normal people.

Abnormalities of a wave

These are either absence of *a* wave or exaggerated *a* waves. *Absence of a wave* is a feature in atrial fibrillation as there is no effective atrial contraction. Absence of a wave also occurs in sinus rhythm immediately after electrical conversion of AF to sinus rhythm as a temporary phenomenon till mechanical activity of the atrium is restored.

In the presence of sinus rhythm with the normal sequence of atrial and ventricular contraction, a *prominent a wave* occurs when the tricuspid valve is obstructed as in tricuspid stenosis, or when the right atrium contracts against a

THE JUGULAR VENOUS PULSE

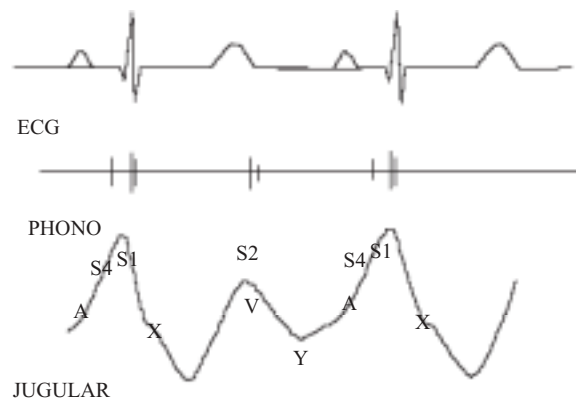


Fig. 14.4: JVP – prominent 'A' wave

non-compliant right ventricle as in a severe concentric right ventricular hypertrophy (Fig 14.4). Concentric hypertrophy of right ventricle occurs in either significant pulmonic stenosis or pulmonary arterial hypertension. In the absence of pulmonic stenosis or pulmonary arterial hypertension, it may be due to right ventricle cardiomyopathy or severe septal hypertrophy as in asymmetric septal hypertrophy. Acute reduction in right ventricle compliance may also occur as in right ventricle infarction in association with inferior myocardial infarction. In the presence of severe aortic regurgitation and a collapsing arterial pulse, the transmitted *c* wave may be sharp and may simulate a prominent *a* wave.

When the sequence of atrial and ventricular contraction is disturbed as in atrioventricular dissociation due to any cause (such as, complete heart block, classic AV dissociation or ventricular tachycardia), the atrium may contract against closed tricuspid valve. Normally, the main force of atrial systole is expended in distending the ventricle and part of the atrial energy is wasted away into the neck veins as a wave. (The main purpose of the atrium is filling the ventricle and not production of an *a* wave!) In situations where the tricuspid valve is closed at the time of atrial contraction, all the atrial energy is 'wasted' away into the neck veins as a very prominent *a* wave and is called the *cannon wave*. In all the conditions with A-V dissociation the P-R interval varies unexpectedly and irregular cannon waves occur. But when the atrium and ventricle are simultaneously activated by a focus in the A-V junction as in junctional rhythm, a regular cannon wave occurs. When the P-R is longer than 0.21 seconds, (first degree heart block), the *a* wave can be appreciated well in advance of first sound.

The causes of a prominent *a* wave are resistance to right atrial emptying at tricuspid and right ventricle level.

- Tricuspid level
 - Tricuspid stenosis
 - Right atrial tumours
- Right ventricular level: Severe concentric right ventricular hypertrophy due to
 - Severe pulmonary hypertension with intact interventricular septum
 - Right ventricular cardiomyopathy
 - Asymmetric septal hypertrophy as in hypertrophic cardiomyopathy
 - Severe aortic stenosis with septal hypertrophy as part of symmetric left ventricular hypertrophy
 - Acute pulmonary embolism
 - Acute tricuspid regurgitation

The causes of cannon waves are:

Regular cannon waves (Fig. 14.5)

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- Junctional rhythm
- Ventricular tachycardia 1:1 retrograde conduction
- Isorhythmic AV dissociation

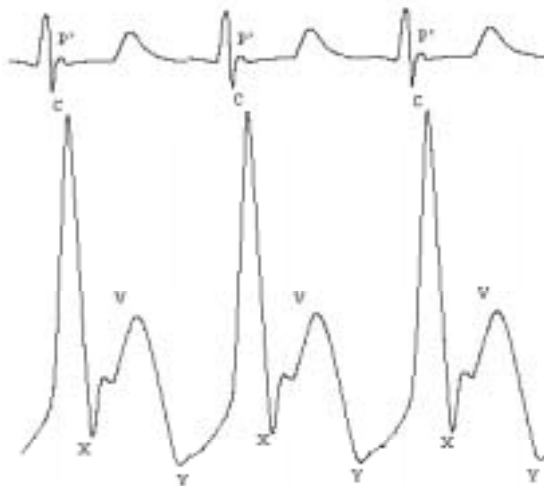


Fig. 14.5: JVP – regular cannon *a* waves: Regular cannon waves (C) are seen with junctional rhythm with 1:1 retrograde atrial activation (P')

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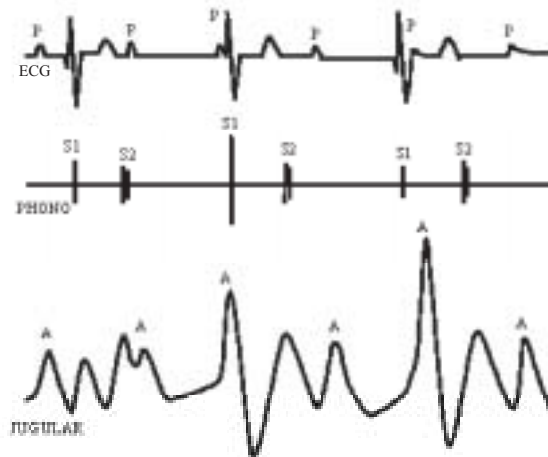


Fig. 14.6: JVP – irregular cannon a waves: Irregular cannon waves associated with complete heart block. Corresponding first heart sound (S1) is louder due to short P-R interval

Irregular cannon waves (Fig. 14.6)

- Complete heart block
- Ventricular tachycardia
- Ventricular ectopy
- Ventricular pacing
- Classic AV dissociation

The causes of absent *a* waves are:

- Atrial fibrillation
- Post-DC conversion of atrial fibrillation
- Sinoventricular conduction in hyperkalemia

The situations in which a single wave occurs are:

- Heart rates above 120/minute
- Severe tricuspid regurgitation with obliteration of x descent
- Chronic tricuspid regurgitation
- Acute tricuspid regurgitation

When the heart rates are beyond 120/minute, *a* and *v* waves merge together to produce a single wave and the details of wave pattern are difficult to discern. The *a* and *v* will also be difficult differentiate one from each other when the x descent is obliterated as in early *v* wave of severe chronic tricuspid regurgitation or in

acute tricuspid regurgitation due to infective endocarditis. In the latter situation the heart rates are often beyond 120/min.

Abnormalities of x descent

Normally during x descent, the atrial pressure is falling due to relaxation of the atrium and also descent of the atrioventricular septum. If for some reason the atrial pressure fails to fall, the x descent gets obliterated (Table 14.4). If there is no atrial contraction, there is generally no x descent, as in atrial fibrillation. As the descent of atrioventricular septum also contributes to x descent, occasionally an x descent may be seen or recorded in spite of atrial fibrillation. This is more likely in patients with constrictive pericarditis dominantly involving the atrioventricular groove. The x descent may be obliterated in tricuspid regurgitation. The influencing factors are severity of tricuspid regurgitation, size of the right atrium and whether the tricuspid regurgitation is acute or chronic. The more severe the tricuspid regurgitation, the smaller the RA, the earlier the v wave obliterates the x descent. In acute tricuspid regurgitation, in addition to the above features there is sinus tachycardia above 120/minute, merging the a wave with the v wave. An exaggerated x descent may be seen in constrictive pericarditis.

Abnormalities of v wave

The v wave reflects the filling pattern of the right atrium and depends on the venous return through the venae cavae. If there is any exaggerated filling of right atrium as in all conditions where the venous pressure is high (such as, right ventricle failure or constrictive pericarditis) or when there is more than one source for the right atrium to fill, as in tricuspid regurgitation from the right ventricle, ASD from the left atrium and ASD with mitral regurgitation when the left atrial systolic v wave is transmitted into the right atrium and neck veins, then the v wave will be prominent.

Table 14.4: Abnormal x descent

Absent x descent	Atrial fibrillation Severe chronic tricuspid regurgitation Acute tricuspid regurgitation Constrictive pericarditis
Prominent x descent	Cardiac tamponade

THE JUGULAR VENOUS PULSE

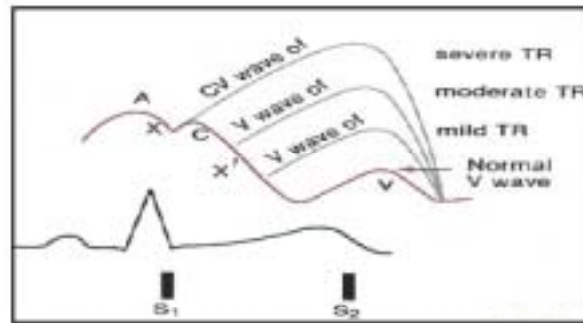


Fig. 14.7: Ventricularisation of *v* wave with obliteration of *x* descent due to severe tricuspid regurgitation

The most prominent *v* waves occur in tricuspid regurgitation with a surging and expansile wave in the neck. The prominent *v* wave in the neck is accompanied by a systolic impulse over the liver. The presence, absence and amplitude of the *v* wave depends on the severity of tricuspid regurgitation, and the size of the right atrium. A mild tricuspid regurgitation may exist without any significant change in right atrial pressure and thereby no *v* wave may occur. Significant tricuspid regurgitation may occur into a large accommodative right atrium without much alteration in right atrial pressure (Fig. 14.7). Lesser degrees of tricuspid regurgitation may produce significant rise in right atrial pressure and prominent *v* wave when the right atrium is relatively small and non-accommodative. With high right ventricle pressure and severe tricuspid regurgitation, the systolic right ventricle pressures may be transmitted across the peripheral veins and may produce unusual phenomena like pulsatile exophthalmos with each *v* wave and palpable venous pulse simulating a collapsing pulse at the brachial or femoral pulses. In such states the *v* wave may measure as much as 60 mmHg and is difficult to obliterate in the neck.

Table 14.5: Abnormal *v* waves

Prominent <i>v</i> wave	<ul style="list-style-type: none"> Right ventricular failure Tricuspid regurgitation Atrial septal defect Atrial septal defect with mitral regurgitation Ventricular septal defect of left ventricle to right atrial communication (Gerbode's defect)
Diminished <i>v</i> wave	<ul style="list-style-type: none"> Hypovolemia Venodilators (nitrates)

Abnormalities of y descent

During y descent the atrial pressures are falling and the ventricular pressure is rising. All abnormalities related to atrial emptying or ventricular filling should naturally influence the y descent (Table 14.6). The y descent is more rapid than normal in all conditions where the preceding v wave is prominent as in tricuspid regurgitation, heart failure or constrictive pericarditis (Fig. 14.8). A slower y descent occurs in tricuspid stenosis due to the difficulty in atrial emptying. The y descent is reduced or absent in pericardial tamponade as the high intrapericardial pressure does not allow ventricular filling. The auscultatory equivalent of a rapid y descent in the neck is a right sided third heart sound, of the slow y descent the tricuspid diastolic murmur and of no y descent, absent third heart sound in spite of elevated jugular venous pressure as in pericardial tamponade. The slow y descent of tricuspid stenosis is not easily detected by the bedside, as this is a difficult sign to quantify. In patients with elevated JVP and prominent v wave often the most impressive feature in the JVP is the rapid y descent. An unimpressive y descent in the face of elevated jugular venous pressure is suggestive of slow y descent (Fig. 14.9).

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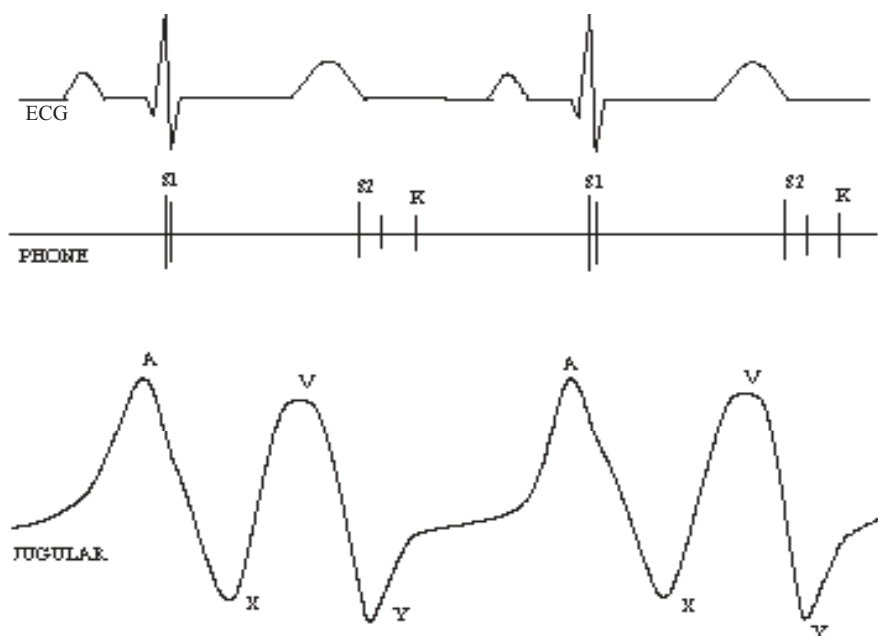


Fig. 14.8: JVP – constrictive pericarditis. JVP wave pattern showing prominent x and y descents. Both a and v wave are prominent, and are equal in height.

THE JUGULAR VENOUS PULSE

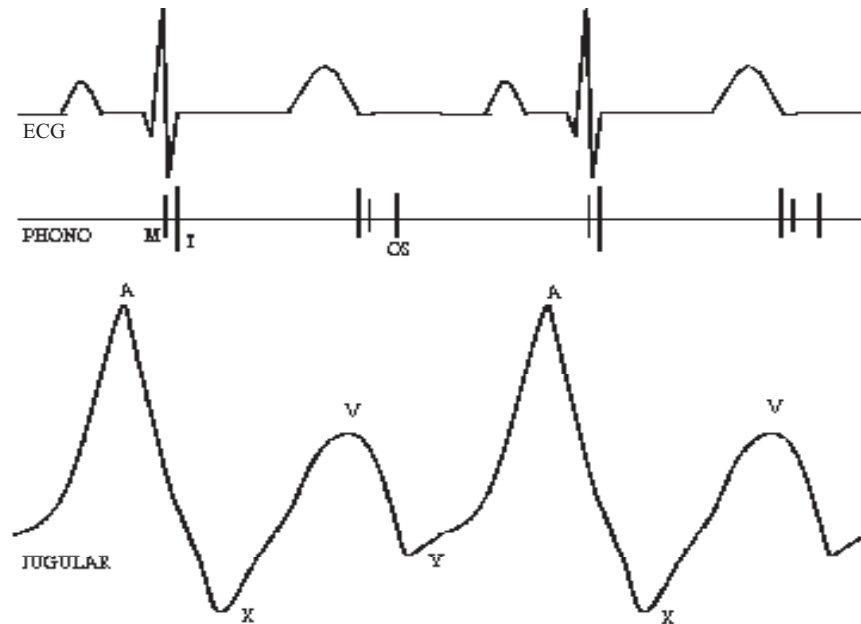


Fig. 14.9: JVP – tricuspid stenosis. JVP wave pattern showing prominent *a* waves and slow *y* descent.

Venous hum

The normal flow of blood across normal veins in the neck is noiseless. However, when there is increased velocity of flow (hyperkinetic states such as thyrotoxicosis) or diminished viscosity of blood (as in anemia) there occurs a continuous bruit over the neck veins. This is called venous hum (Fig. 14.10). In the supine position, with the veins distended there is little or no turbulence, and therefore no hum is heard.

Table 14.6: Abnormal *y* descent

Rapid <i>y</i> descent (Correlates with RV S3)	<ul style="list-style-type: none"> All causes of prominent <i>v</i> wave Right ventricular failure Tricuspid regurgitation Constrictive pericarditis Atrial septal defect Atrial septal defect with mitral regurgitation Ventricular septal defect of LV to RA type
Slow <i>y</i> descent (Absent S3)	<ul style="list-style-type: none"> Tricuspid stenosis Pericardial tamponade (<i>y</i> descent may be absent) Tension pneumothorax

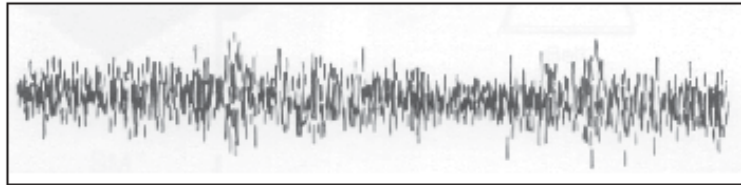


Fig. 14.10: Continuous murmur of venous hum

In a sitting patient with the bell of the stethoscope lightly applied at the base of the neck between the two heads of the sternomastoid, the venous hum can be heard as a continuous murmur. The hum disappears by interrupting the venous flow (by pressure above the stethoscope). The venous hum is normally heard in the majority of children and also during pregnancy. The presence of a venous hum in adults, is generally abnormal. It occurs in hyperkinetic circulatory states like anemia or thyrotoxicosis. It also occurs in intra cranial or head and neck arteriovenous fistulas. If a venous hum is heard in an adult in the absence of anemia, one should check for thyrotoxicosis. In a child with congestive heart failure and a high volume arterial pulse, look for a bruit over the head and a venous hum in the neck to rule out an intracranial arteriovenous fistula.

The causes could be physiological or pathological (Table 14.7).

An audible venous hum in the presence of congestive heart failure should suggest the possibility of hyperkinetic state like severe anemia or thyrotoxicosis with heart failure. Intra cranial arteriovenous fistula is a rare but important cause of heart failure in infancy. A cervical venous hum with high volume arterial pulse and bruit over the skull suggest the diagnosis.

Table 14.7: Causes of venous hum

Physiological	Children Pregnancy Normal adults (rarely)
Pathological	Hyperkinetic states Anemia Thyrotoxicosis Beriberi Intracranial arteriovenous fistula Compression of jugular vein by fascia, or bony structures in the neck

Respiratory variation

Normally during inspiration venous return to the right side of the heart increases. However, this is accommodated by the inspiratory decrease in pulmonary vascular impedance. As a result the pulmonary artery, right ventricle and right atrial pressures fall in spite of increased venous return. During expiration the lungs are squeezed of their air and the pulmonary circulation is compressed by the thoracic cage increasing the pulmonary impedance and pressures. As a result the venous column rises during expiration and falls during inspiration. In conditions where the increase in venous return of inspiration cannot be translated as more pulmonary filling, inspiratory filling of neck veins occurs. This is called Kussmaul's sign and occurs in constrictive pericarditis, restrictive cardiomyopathy, tricuspid stenosis, or severe right ventricular failure itself. In many of these patients the venous column is beyond the angle of the jaw and is not easily available. In these conditions with inspiration, the wave pattern appears exaggerated while there is no obvious and discernible change in venous pressure as it is already very high. Conversely with expiration the venous wave undulations appear diminished.

Hepatojugular reflux

When the venous return is increased to the right ventricle, it readily accommodates it and translates it as more output into the lungs and there is only a transient rise in pressure. Abdominal compression increases venous return further and results in only a transient rise in jugular venous pressure with the right ventricle emptying the veins in the next few beats. In the presence of right ventricular dysfunction, abdominal compression results in a longer duration of pressure rise (Fig. 14.11).

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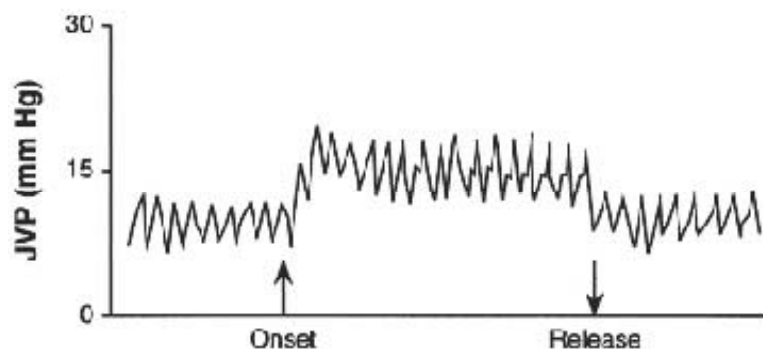


Fig. 14.11: Increase in RA pressure with abdominal compression: hepatojugular reflux

As a guideline a 10 second abdominal compression results in rise in JVP that lasts less than 10 seconds; if it persists for more than 15 seconds it is suggestive of right ventricle dysfunction. It may be the earliest sign to appear in right ventricle dysfunction, occult constrictive pericarditis or tricuspid stenosis. Recent studies demonstrated a good correlation between positive abdomino-jugular reflux and increased pulmonary capillary wedge pressures (Fig. 14.12).

Liver pulsations

The liver should be considered an extension of the right atrial cavity. It generally reflects the jugular venous pressure rise by enlargement, and jugular venous pulsations like *a*, *v* waves and rapid *y* descent are easily evident over the liver. Due to the further delay of events from the RA to the liver, the pre-systolic wave or *a* wave seem to appear immediately after the first heart sound, and the systolic wave or *v* wave slightly later, after the second heart sound. The *y* descent in the neck and rapid outward filling of the right ventricle in the parasternal region (third heart sound) coincide with inward movement of the liver. In an infant, the liver is the only guide to the recognition of elevated right atrial pressure as the JVP is difficult to delineate.

JVP IN SPECIFIC DISEASES

1. *Jugular venous pulse in superior vena cava obstruction:* In superior vena cava obstruction, the level is elevated usually above the angle of the jaw and the

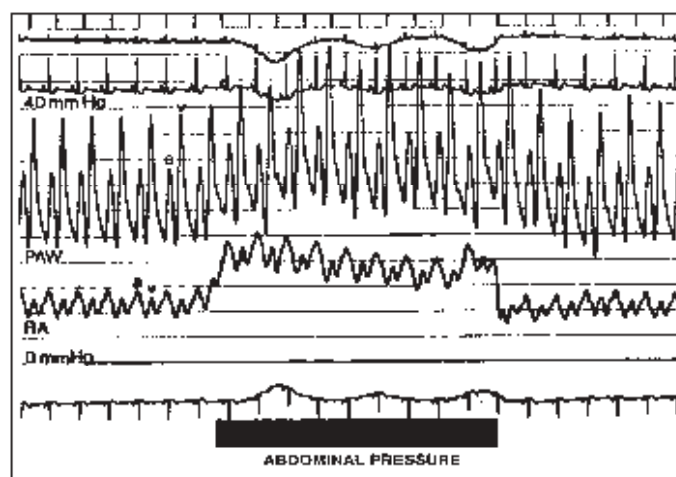


Fig. 14.12: Correlation between abdominal compression and pulmonary wedge pressure

THE JUGULAR VENOUS PULSE

pulsations are absent. The liver is not enlarged. A severely elevated jugular venous pressure without pulsations is often missed, unless one carefully looks for it. The associated edema of the neck makes it more difficult to elicit this sign.

2. Jugular venous pulse in tricuspid stenosis: In tricuspid stenosis, the findings depend upon the severity of tricuspid stenosis, the rhythm, the volume status of the patient (diuretic therapy) and associated lesions (Table 14.8).

Elevated jugular venous pressure in a patient with mitral stenosis, with no significant dyspnea or PND, could indicate associated tricuspid stenosis.

3. Jugular venous pulse in tricuspid regurgitation: The jugular venous pulse appearance in tricuspid regurgitation depends on the severity of tricuspid

Table 14.8: JVP in tricuspid stenosis

<i>Level</i>	Elevated or normal	Obstruction to tricuspid valve. The level of JVP is a reflection of the severity of TS and the volume status. The level may be normal in mild TS or even with moderate TS on diuretics.
<i>Wave pattern</i>		
<i>a wave</i>	Prominent and can reach as high as 30 mmHg equaling RV systolic pressure (giant <i>a</i> wave)	Obstruction to pre-systolic atrial emptying due to tricuspid stenosis. Longstanding TS elicits right atrial hypertrophy and dilatation increasing the force of contraction against narrow tricuspid valve. Most of the atrial energy is wasted away into the neck veins. Pre-systolic hepatic impulse is common.
\times descent	Normal and consistent with large <i>a</i> wave	Obliterated only when atrial fibrillation develops late in the disease
<i>v wave</i>	Normal and consistent with the degree of JVP elevation	Filling of obstructed RA will result in larger <i>v</i> wave than normal, but is always less than <i>a</i> wave. Once AF sets in, <i>v</i> wave may be the only positive wave
<i>y descent</i>	Slow <i>y</i> descent	Obstruction to right atrial emptying. Rapid <i>y</i> descent excludes TS, but this sign is unreliable in actual practice

Table 14.9: Jugular venous pulse in tricuspid regurgitation

<i>Feature</i>	<i>Findings</i>	<i>Mechanisms/ significance</i>
<i>Level</i>	Normal or elevated	Right ventricular failure
<i>Wave pattern</i>		
<i>a wave</i>	Prominent with PAH or acute organic TR. <i>a</i> wave may merge with <i>v</i> wave with severe tricuspid regurgitation	Severe PAH with non-compliant RV
\times descent	Preserved with milder or chronic TR or larger RA Obliterated with severe TR and smaller RA or acute TR	Obliteration of \times descent occurs with severe TR or smaller RA or acute TR
<i>v wave</i>	Can be very prominent or normal	Large <i>v</i> wave with severe TR, Smaller RA and acute TR. Small or no <i>v</i> wave with milder TR, larger RA or chronic TR
<i>y</i> descent	Steep and impressive Slow with associated TS	Rapid filling of RV Slower filling of RV
<i>h wave</i>	Can be prominent, can be mistaken for <i>a</i> wave	Occurs after the rapid filling wave in RV and may be reflected into RA

regurgitation, the size of right atrium, the systolic pressure in right ventricle, and the mode of onset of tricuspid regurgitation (acute or chronic) (Table 14.9).

The more severe abnormalities are seen with more severe tricuspid regurgitation, smaller right atrium, and higher right ventricle systolic pressure.

The H wave can be mistaken for an *a* wave, and may be the source for confusion in patients with atrial fibrillation.

4. Jugular venous pulse in pulmonic stenosis: The findings depend on the severity of pulmonic stenosis, the presence or absence of right ventricular failure, and the associated lesions (Table 14.10).

Pure pulmonic stenosis is often mistaken for small ventricular septal defect and tetralogy of Fallot. The jugular venous pulse is of help in this setting. If jugular venous pressure is elevated and the *a* wave is prominent; it rules out small ventricular septal defect and tetralogy.

5. Jugular venous pulse in mitral stenosis: The JVP in mitral stenosis depends on the severity of mitral stenosis, degree of pulmonary arterial hypertension, associated organic tricuspid valve disease, diuretic therapy, rhythm (atrial fibrillation

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or sinus rhythm) and the presence or absence of right ventricular failure. Mild mitral stenosis with no pulmonary arterial hypertension and sinus rhythm will show normal jugular venous pressure (Table 14.11).

Table 14.10: Jugular venous pulse in pulmonary stenosis

<i>Features</i>	<i>Findings</i>	<i>Mechanisms/ significance</i>
<i>Level</i>	Normal or elevated with right ventricular failure	Elevated JVP usually means RV systolic dysfunction
<i>Wave pattern</i>		
<i>a wave</i>	Prominent with severe PS can be 'jumping'	Elevated JVP favours intact ventricular septum, tetralogy is unlikely.
\times descent	Normal, or may be obliterated with severe TR	Hypertrophied, small cavities, non-compliant right ventricle
<i>v wave</i>	Normal, may be increased with Right ventricular failure	Severe TR results in early filling of RA
<i>y descent</i>	Normal, or rapid with right ventricular failure or TR	Increased atrial filling

Table 14.11 Jugular venous pulse in mitral stenosis

<i>Features</i>	<i>Findings</i>	<i>Mechanisms/ significance</i>
<i>Level</i>	Normal or elevated	Elevated with Right ventricular failure Associated organic tricuspid disease Associated atrial septal defect (Lutembacher syndrome)
<i>Wave pattern</i>		
<i>a wave</i>	Normal or prominent	Prominent <i>a</i> wave with Tight mitral stenosis with severe PAH Associated TS Associated ASD
\times descent	Normal or obliterated	Absent with Atrial fibrillation Severe TR
<i>v wave</i>	Normal or prominent	Prominent with Right ventricular failure TR
<i>y descent</i>	Normal or rapid or slow	Rapid with right ventricular failure, TR Slow with associated TS

The most impressive of jugular venous pulse features are seen with severe mitral stenosis, severe pulmonary arterial hypertension, severe functional tricuspid regurgitation and right ventricular failure. The *v* wave in some patients can be so prominent that it is mistaken for an arterial pulse. Rarely, the *v* wave can be transmitted to the more peripheral pulses veins like femoral veins. The earlobe may move with each *v* wave and pulsatile exophthalmos may be seen in some patients with severe tricuspid regurgitation.

6. Jugular venous pulse in mitral regurgitation: The jugular venous pressure in mitral regurgitation depends on the presence or absence of pulmonary arterial hypertension, size of the left atrium, right ventricular failure, cause of mitral regurgitation, rhythm of the heart, associated organic tricuspid valve disease or

Table 14.12: Jugular venous pulse in mitral regurgitation

<i>Feature</i>	<i>Finding</i>	<i>Mechanism/ significance</i>
<i>Level</i>	Normal or elevated	Elevated JVP with PAH and right ventricular failure Associated ASD Organic TVD RV infarct in mitral regurgitation of CAD Secondary MR with myocardial dysfunction of cardiomyopathy/CAD
<i>Wave pattern</i> <i>a</i> wave	Normal or prominent	Prominent <i>a</i> wave with Severe pulmonary hypertension with mitral regurgitation Severe PAH with TR mistaken for mitral regurgitation Mitral regurgitation in association with HOCM Inferior MI, mitral regurgitation due to PMD and associated right ventricular infarction
<i>x</i> descent	Normal or obliterated	May be obliterated with mitral regurgitation associated with ASD
<i>v</i> wave	Normal or prominent	Prominent with Pulmonary hypertension and right ventricular failure Associated tricuspid regurgitation
<i>y</i> descent	Normal or prominent	Associated atrial septal defect Rapid <i>y</i> descent with any of the above conditions

ASD. For a particular degree of mitral regurgitation, the size of the left atrium influences the degree of pulmonary venous hypertension, which in turn is the mediator for pulmonary arterial hypertension and the consequent right ventricular failure (Table 14.12).

In the most common form of mitral regurgitation (rheumatic mitral regurgitation), prominent *a* wave in the neck veins are always associated with either severe pulmonary arterial hypertension, or organic tricuspid stenosis. In mitral regurgitation associated with hypertrophic obstructive cardiomyopathy, and papillary muscle dysfunction of inferior myocardial infarction with associated right ventricular infarction, the *a* wave may be prominent without accompanying pulmonary arterial hypertension. Though the severity of mitral regurgitation has no direct influence on JVP, the degree of pulmonary arterial hypertension influences it. The degree of pulmonary arterial hypertension is in turn related to the severity of mitral regurgitation and the size of the left atrium. A smaller left atrium even with a moderate mitral regurgitation, results in significant pulmonary venous and arterial hypertension leading to right ventricular failure.

7. Jugular venous pulse in aortic stenosis: The jugular venous pulse in aortic stenosis depends on the severity of aortic stenosis, presence or absence of right ventricular failure, associated mitral valve disease with pulmonary arterial hypertension or organic tricuspid valve disease.

Table 14.13: JVP in aortic stenosis

Feature	Finding	Mechanism/significance
Level	Normal or elevated	Elevated with RVF secondary to LVF Associated mitral stenosis, PAH and RVF Associated organic tricuspid valve disease
Wave pattern <i>a</i> wave	May be prominent	Prominent with HOCM with or without associated RVOT obstruction Any severe AS Associated mitral stenosis + PAH Associated TS
\times descent, <i>v</i> wave and <i>y</i> descent	Normal	May be abnormal with RVF, TR, coexisting TS

The elevated jugular venous pressure in aortic stenosis usually occurs late in the disease and carries a poor prognosis. Prominent *a* wave can occur in isolated AS without any pulmonary arterial hypertension due to severe septal hypertrophy decreasing right ventricular compliance as interventricular septum is shared by both ventricles. In 'fixed' obstruction, a prominent *a* wave always indicates severe aortic stenosis in the absence of associated lesions. If the *a* wave is prominent with 'mild aortic stenosis', consider hypertrophic cardiomyopathy, associated mitral stenosis with pulmonary arterial hypertension, or associated tricuspid stenosis. Late in the disease with severe right ventricular failure and elevated JVP, the severity of AS may be underestimated.

8. Jugular venous pulse in aortic regurgitation: The jugular venous pressure in aortic regurgitation depends on the severity, presence or absence of right ventricular failure, associated lesions at the mitral or tricuspid valve and the cause of aortic regurgitation (Table 14.14). The commonest error in actual practice is to mistake the *c* wave of carotid artifact as prominent *a* wave.

9. Jugular venous pulse in atrial septal defect: The determinants of jugular venous pressure in atrial septal defect are pulmonary arterial hypertension, associated left sided disease, pulmonic stenosis, right ventricular function, and the

Table 14.14: Jugular venous pulse in aortic regurgitation

Feature	Significance/ mechanism
Level	Normal or elevated Elevated with RVF secondary to LVF Associated tricuspid valve disease Bernheim's syndrome Aneurysm of ascending aorta Dissection of ascending aorta Aortic regurgitation with CRF and fluid load
Wave pattern <i>a</i> wave	Normal, rarely prominent Prominent with Associated TS Associated mitral stenosis and PAH Prominent <i>c</i> wave due to carotid pulse simulating <i>a</i> wave
x descent, <i>v</i> wave, <i>y</i> descent	Depend on presence of RVF and associated lesions
Venous hum	May be mistaken for the murmur of AR

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cardiac rhythm. Usually *a* wave and the *v* wave tend to be equal in height but in some patients the *v* may be slightly more prominent. Similarly expected slight fall in venous pressure with inspiration may not be obvious due to phasic alterations in left to right shunt.

Evaluation of jugular venous pulse in atrial septal defect gives important clues as to the presence or absence of one these conditions (Table 14.15).

Table 14.15: Jugular venous pulse in atrial septal defect (ASD)

<i>Feature</i>	<i>Finding/ significance</i>
<i>Level</i> Elevated	Usually normal Mitral valve disease (Lutembacher syndrome) Left ventricular failure of any cause Severe PAH with RVF RVF due to volume load Underlying TAPVC
<i>Wave pattern</i> <i>a</i> wave	Normal Prominent with Associated mitral stenosis PAH PS
<i>x</i> descent	Normal
<i>v</i> wave	Prominent due to overfilling of RA from venae cavae and LA PAH with TR Associated mitral regurgitation
<i>y</i> descent	Prominent with any of the above

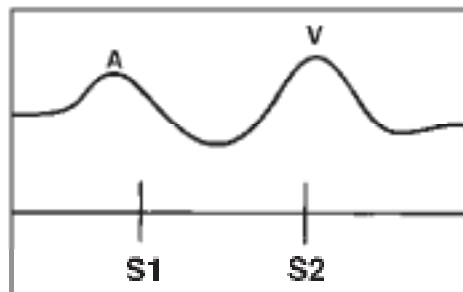


Fig. 14.13: JVP in ASD; note the prominent *v* wave

In an isolated secundum atrial septal defect without pulmonary arterial hypertension, the jugular venous pulse as a rule is not elevated and the *v* wave is slightly prominent (Fig. 14.13). If the jugular venous pressure is elevated in atrial septal defect with continuing left to right shunt, one should consider associated mitral valve disease.

10. Jugular venous pulse in ventricular septal defect: In small to moderate ventricular septal defects, the jugular venous pressure is normal. In large ventricular septal defect with heart failure in infancy, the jugular venous pressure may be elevated. In spite of severe pulmonary arterial hypertension, the *a* wave is generally not prominent. In an infant, the jugular venous pulse is not easy to evaluate and the liver size and pulsations are of use. In a patient with a long systolic murmur, the presence of a prominent *a* wave supports the possibility of pulmonic stenosis over ventricular septal defect (Table 14.16).

Elevated jugular venous pressure or congestive heart failure in ventricular septal defect is a feature of large ventricular septal defect in infancy and is rare in older children. If the jugular venous pressure is elevated in an older child or adolescent, left ventricle to right atrial defect or tricuspid regurgitation due to cleft leaflet (part of AV canal defect), or due to infective endocarditis should be considered.

Table 14.16: Jugular venous pulse in ventricular septal defect (VSD)

<i>Feature</i>	<i>Finding / significance</i>
<i>Level</i>	Normal with small VSD
Elevated	Large VSD with CCF in infancy VSD with MR/TR as in AV canal defects LV to RA communication (Gerbode's defect) Associated large PDA, severe AR
<i>Wave pattern</i>	
<i>a</i> wave	Normal Prominent with Severe PS mistaken for VSD Restrictive VSD with severe PS
\times descent	Normal Obliterated with TR, LV to RA defect
<i>v</i> wave	Normal Prominent with CCF, TR, LV to RA defect
<i>y</i> descent	Normal Prominent with <i>v</i> wave prominence

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Table 14.17: Jugular venous pulse in Eisenmenger syndrome

<i>ASD</i>	<i>VSD</i>	<i>PDA</i>
Level may be elevated	Usually normal	May be elevated
<i>a</i> wave normal; absent with AF	Normal	May be prominent
<i>v</i> wave prominent with CCF or TR	Normal, CCF and TR rare	As in ASD

11. Jugular venous pulse in Eisenmenger syndrome: The jugular venous pulse in Eisenmenger syndrome gives clues to the underlying defect (Table 14.17).

In primary pulmonary arterial hypertension with right to left shunt at atrial level, the *a* wave is usually prominent and the JVP is often elevated by the time cyanosis appears. In atrial septal defect and ventricular septal defect, the *a* wave is not prominent in spite of severe pulmonary arterial hypertension due to the underlying defects permitting easy right atrial decompression. In ventricular septal defect with Eisenmenger syndrome, elevated JVP is rare unlike the other two defects.

12. Jugular venous pulse in congenital cyanotic heart disease: Evaluation of jugular venous pulse gives valuable clues to the underlying defect in this set of disorders (Table 14.18). Tetralogy of Fallot, or lesions with similar physiology (non-restrictive ventricular septal defect with significant pulmonic stenosis) have unremarkable jugular venous pulse with normal level and wave pattern.

Elevated jugular venous pressure in cyanotic heart disease usually means either an intact ventricular septum or increased pulmonary flow or both. Rarely the

Table 14.18: Jugular venous pulse in cyanotic heart disease

<i>TOF or TOF-like physiology</i>	<i>PS with intact IVS + Right to left atrial shunt</i>	<i>Tricuspid atresia</i>	<i>Transposition of arteries or veins with high pulmonary flow (TGA, TAPVC)</i>
Level is normal	Usually elevated	Elevated with restrictive ASD	Usually elevated
Wave pattern			
<i>a</i> wave normal	Prominent	Prominent	May be prominent
<i>v</i> wave normal	Normal	Normal	Normal
<i>y</i> descents vary by the waves	Prominent with TR	Prominent with MR	Prominent with CCF or TR

Table 14.19: Causes of elevated jugular venous pressure in tetralogy of Fallot

<i>Cause</i>	<i>Mechanism</i>
Anemia	Volume load
Systemic hypertension	Non-compliant ventricles Biventricular failure
Infective endocarditis	Aortic regurgitation Tricuspid regurgitation Anemia Renal failure
Aortic regurgitation	Volume load
Cardiomyopathy	Ventricular dysfunction
Bronchopulmonary collaterals	Volume load Increased pulmonary flow
After systemic to pulmonary shunt	Volume load Increased pulmonary flow
Associated PDA	Volume load
Adult tetralogy	Myocardial dysfunction

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jugular venous pressure may be elevated in tetralogy of Fallot due to an associated abnormality (Table 14.19).

The wave pattern as mentioned earlier is normal in tetralogy of Fallot. However, the *a* wave may be prominent in adult tetralogy, tetralogy of Fallot with restrictive ventricular septal defect, systemic hypertension, or cardiomyopathy.

The causes of a prominent a wave in tetralogy of Fallot are:

- Adult tetralogy
- Restrictive ventricular septal defect
- Systemic hypertension
- Cardiomyopathy

As a general rule, some other disorder should be considered when an *a* wave is prominent. Consider the following conditions when there is a prominent *a* wave in cyanotic heart disease:

- Tricuspid atresia
- Pulmonic stenosis with intact ventricular septum and right to left atrial shunt
- Total anomalous venous connection

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- Pulmonary atresia with intact ventricular septum
- Adult tetralogy
- TOF with systematic hypertension
- TOF with restrictive VSD
- TOF with cardiomyopathy

In infants, the pulsations of the liver are more often used than the jugular venous pulse and the *a* wave appears to occur along with or immediately after the first heart sound.

The venous hum is uncommon in congenital cyanotic heart disease. When present, it should raise the possibility of total anomalous pulmonary venous connection into the superior vena cava, severe anemia or hepatic cirrhosis with multiple microvascular pulmonary arteriovenous fistulae.

13. Jugular venous pulse in cardiomyopathies The jugular venous pulse in cardiomyopathies depends on whether the cardiomyopathy is dilated or restrictive (Table 14.20).

It is often mistakenly believed that dilated or congestive cardiomyopathy always has congestive heart failure or elevated jugular venous pressure. Congestive cardiomyopathy can exist with or without congestive heart failure or elevated jugular venous pressure. In obliterative cardiomyopathy, the *a* wave is often prominent and is lost after the onset of atrial fibrillation. With severe tricuspid regurgitation, the *v* wave assumes prominence. Selective prominence of either *a* or *v* waves distinguish cardiomyopathies from constrictive pericarditis.

14. Jugular venous pulse in pericardial disease: The jugular venous pulse in pericardial disease depends on the clinical syndrome. In pericardial effusion without

Table 14.20: Jugular venous pulse in cardiomyopathy

Feature	Dilated CM	Restrictive	Obliterative (EMF)
Level	Normal/elevated	Normal/elevated	Usually elevated
Wave pattern			
<i>a</i> wave	Normal	Prominent	Prominent
<i>v</i> wave	Normal/prominent	Normal	Prominent (TR)
\times descent	Normal	Normal	May be obliterated with TR
<i>y</i> descent	Normal/prominent	Normal	Prominent

Table 14.21: Jugular venous pulse in pericardial tamponade

<i>Feature</i>	<i>Finding</i>	<i>Mechanism</i>
<i>Level</i>	Marked elevation usually above angle of jaw	Cardiac compression due to high intrapericardiac pressure
<i>Wave pattern</i>		
<i>a wave</i>	Never prominent	The atrial compression prevents exaggerated atrial contraction
\times descent	Normally preserved	Fall in atrial pressure due to ventricular contraction producing descent of atrioventricular septum
<i>v wave</i>	Normal	Atrial filling is preserved
<i>y descent</i>	Obliterated or absent	Ventricular compression due to high intrapericardiac pressure prevents rapid ventricular filling
Kussmaul's sign	May be present	Additional venous return not admissible due to cardiac compression

cardiac compression, the JVP is not elevated. A mere elevation of JVP is not suggestive of cardiac tamponade. In cardiac tamponade, the JVP is elevated usually above the angle of the jaw. The *a* and *v* waves are equal and the *y* descent is obliterated or absent. The \times descent is more prominent.

The jugular venous pressure as a rule, is grossly elevated in cardiac tamponade. However, in low pressure cardiac tamponade, it may not be elevated due to accompanying hypovolemia. As pericardial tamponade is not considered as a diagnosis in the absence of elevated jugular venous pressure, one must be aware of situations when cardiac tamponade occurs without elevated jugular venous pressure.

The causes of cardiac tamponade without elevated jugular venous pressure (low pressure tamponade) are given in Table 14.22.

As a general rule, over a stable preexisting pericardial effusion, hypovolemia precipitates tamponade in the above situations.

Though cardiac tamponade and constrictive pericarditis are pericardial disorders, the jugular venous pressure is different in both these conditions, and gives clues to differential diagnosis (Table 14.23).

The most impressive sign in jugular venous pulse in constriction is rapid *y* descent with equal *a* and *v* waves. If there is a very prominent *a* wave or *v* wave, constriction is unlikely; check for another diagnosis.

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Table 14.22: Causes of low pressure tamponade

<i>Cause</i>	<i>Mechanism</i>
Peri-operative tamponade	Fall in venous pressure due to venodilatation of anaesthesia, blood loss
Postoperative tamponade	Hypovolemia
During hemodialysis	Hypovolemia, bleeding into pericardium due to heparinization
Pericardial tamponade with multiple trauma	Blood loss
Selective left ventricular tamponade and after cardiac surgery	Isolated compression of left ventricle by hematoma, etc.
Diuretic therapy	Hypovolemia

Table 14.23: Jugular venous pulse in constrictive pericarditis

<i>Feature</i>	<i>Finding</i>	<i>Mechanism</i>
<i>Level</i>	Elevated, the severity of elevation is related to the severity of constriction	Variable degree of cardiac compression, the mildest form being occult constriction where the disorder is evident only after fluid load rapidly
<i>Wave pattern</i>		
<i>a wave</i>	Never prominent	Atrial constriction does not permit exaggerated atrial contraction
<i>x descent</i>	May be normal or exaggerated	Constriction around the AV groove results in excessive descent of the AV septum
<i>v wave</i>	Normal, usually equal to <i>a wave</i>	Venous return to right atrium unaffected
<i>y descent</i>	Rapid or steep	Rapid ventricular filling due to active ventricular relaxation encouraged by spring like pericardium. The rapid <i>y</i> descent coincides with diastolic outward movement of the precordium and pericardial knock
Kussmaul's sign	Positive	Additional venous return of inspiration is not accommodated by RV due to rigid shield of constriction

Table 14.24: Elevated jugular venous pressure with shock

<i>Cause</i>	<i>Mechanism</i>
Heart failure	Ventricular or valvular dysfunction
Cardiac tamponade	Cardiac compression
Right ventricular infarction	Right ventricular failure Inadequate filling of left ventricle
Acute pulmonary embolism	Obstruction to pulmonary circulation
Tension pneumothorax	Cardiorespiratory embarrassment
Massive pleural effusion especially bilateral	Cardiorespiratory embarrassment

15. Jugular venous pulse in shock: The findings in jugular venous pulse are often the first clue to the differential diagnosis of shock (Table 14.24). Though a cardiogenic cause is the most likely when the jugular venous pressure is elevated, other non-cardiac conditions can present similarly.

In actual practice, tension pneumothorax and pleural effusion are often not considered in the differential diagnosis of shock.

16. Jugular venous pulse in arrhythmias: In the interpretation of cardiac rhythm, the sequence of atrial to ventricular activity is of primary importance. The *a* and *v* waves in jugular venous pulse correlate with the P wave and the QRS complex in the electrocardiogram. The normal sinus rhythm is characterized by sequential *a* and *v* waves. The *a* wave occurring along with the first sound is a reflection of normal PR interval. The number of *a* waves and *v* waves is equal. Any disturbance in this relationship is suggestive of a different rhythm. Cardiac rhythm can be recognised by the sequence of atrial to ventricular activity, the rate, the presence or absence of cannon waves and the correlation with auscultatory and arterial pulse features. The value of these features are given in Table 14.25.

In a rapid regular tachycardia with wide QRS complexes, the diagnosis could be either ventricular tachycardia (VT) or supraventricular tachycardia (SVT) with aberration. The presence of irregular cannon waves is virtually diagnostic of VT rather than SVT. The patient should be positioned slightly propped up to bring out the venous waves. Holding breath also helps in appreciation of cannon waves. A recent study by Garratt et al showed that the variability in JVP and first heart sound were highly sensitive and specific signs of ventriculoatrial dissociation during tachycardia. Unlike the first heart sound and JVP, the arterial pulse was not of much value in the diagnosis.

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Table 14.25: Jugular venous pulse in arrhythmias

<i>Rhythm</i>	<i>A to V sequence</i>	<i>Cannon waves</i>	<i>Correlations</i>
Sinus rhythm	<i>a</i> precedes <i>v</i> regularly <i>a</i> occurs along with S1	Absent	S1: Constant S4: Regular
First degree heart block	<i>a</i> precedes <i>v</i> regularly <i>a</i> wave occurs before S1 or even well before	Absent Rarely regular cannon waves occur with extreme prolongation of PR, with P wave falling on the preceding T wave	S1: Diminished S4: S4-S1 interval long
Wenckebach's phenomenon	Gradual lengthening of A-V interval, ends with <i>a</i> wave that is not followed by <i>v</i> or S1	Absent	S1: Progressive diminution in intensity with pause
Mobitz type II block	Constant A-V interval, suddenly <i>a</i> is not followed by <i>v</i> .	Absent	Constant S1 followed by sudden pause 2:1 ratio between <i>a</i> waves and S1
Second degree AV block	Two <i>a</i> waves for one <i>v</i> wave or S1	Absent. May be present if the non-conducted P wave falls on the preceding T wave. Cannon waves are 2:1.	Constant S1
Complete heart block	Variable A-V interval More <i>a</i> waves than <i>v</i> waves or S1	Present, irregular May be absent with associated AF	S1: Variable S4: Variable Arterial pulse volume may vary
Junctional rhythm	<i>a</i> occurs after S1	Regular	S1: Constant
Ventricular tachycardia	Variable relationship Regular with retrograde atrial activation	Irregular Regular with retrograde atrial conduction	S1: Variable constant with retrograde atrial conduction Arterial pulse may vary in volume
Atrial tachycardia	<i>a</i> to <i>v</i> sequence normal	Absent	S1: Constant Arterial pulse constant in volume

DIAGNOSTIC CLUES IN PRACTICE

- ➔ When checking the JVP there is nothing sacrosanct about the 45 degree angle. The only thing about jugular venous pulse many people remember after leaving medical school is this 45 degrees and nothing else.
- ➔ Nobody's jugular venous pulse can be measured in the supine position.
- ➔ The easiest way to detect a raised jugular venous pressure is to make the patient sit or stand and if the venous column cannot be seen above the upper border of the clavicle, the jugular venous pressure is not elevated.
- ➔ The first heart sound coincides with the *a* wave, and the second heart sound with the *v* wave. The *x* descent follows the first heart sound and the *y* descent follows the second heart sound.
- ➔ Elevated jugular venous pressure with no history of significant dyspnea, paroxysmal nocturnal dyspnea or orthopnea generally indicates *pure right heart failure*.
- ➔ In a patient with mitral stenosis, elevated jugular venous pressure, edema of face or feet without dyspnea, PND, or orthopnea indicates *associated tricuspid stenosis*.
- ➔ Prominent *a* wave, slow *y* descent, no right ventricular impulse, no palpable P2 indicates *tricuspid stenosis*.
- ➔ Elevated jugular venous pressure rapid *y* descent, diastolic outward moment of the whole precordium coinciding with the *y* descent and pericardial knock indicates *constrictive pericarditis*.
- ➔ Elevated jugular venous pressure but no *y* descent or third heart sound, indicates *pericardial tamponade*.
- ➔ Elevated jugular venous pressure in an atrial septal defect with large left to right shunt (tricuspid mid-diastolic murmur) generally indicates *associated mitral valve disease*.
- ➔ Prominent *a* wave, normal *y* descent, sustained right ventricular impulse palpable S4 and pulmonary arterial pulsations and P2 indicates *severe pulmonary arterial hypertension*. The last two components are substituted by a systolic thrill in the pulmonary area in pulmonic stenosis.
- ➔ Elevated jugular venous pressure but no cyanosis in a patient with severe pulmonary arterial hypertension rules out Eisenmenger syndrome. By the time they have right heart failure, severe pulmonary arterial hypertension,

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- they always have cyanosis in Eisenmenger syndrome.
- ➔ 'Pulseless elevation' in level of JVP is often missed.
- ➔ With extreme elevation of jugular venous pressure as in pericardial tamponade or severe constrictive pericarditis, the very elevation of jugular venous pressure may be missed due to unimpressive venous pulsations.
- ➔ Venous hum is a valuable clinical clue to the presence of hyperkinetic circulatory disease in adults.
- ➔ Venous hum in a cyanotic patient points to the diagnosis of hepatic cirrhosis with multiple microvascular pulmonary arteriovenous fistulae.
- ➔ In a febrile patient with small or moderate VSD, elevated JVP usually indicates infective endocarditis of tricuspid valve with TR.
- ➔ An elevated JVP of more than 7 cm or a prominent abdominojugular test have a sensitivity, specificity and predictive accuracy of about 80 per cent for elevated pulmonary wedge pressure.

How to express findings in jugular venous pulse

If the jugular venous pulse is normal, it may be expressed as follows:

JVP, level is normal, wave pattern is normal, hepatojugular reflux is negative, Kussmaul's sign is negative and there is no venous hum.

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If the jugular venous pulse is abnormal, it may be expressed as follows:

JVP, level is 8 cm above sternal angle, *a* wave is prominent, *v* is normal, *y* descent is rapid. Kussmaul's sign is negative and there is no venous hum.

15 The Cardiac Impulse

The apical impulse is the outermost, lowest point of maximal cardiac impulse. It is formed by the region of the left ventricle adjacent to interventricular septum. With each contraction, all the walls of the left ventricle move inward reducing the ventricular size, except the region of the apex which rotates and moves outward lifting the chest wall over it. The normal apical impulse comes out along with the first sound and stays on as an outward movement as long as the ventricle ejects blood into the aorta. The outward movement retracts from the palpating finger well before the second heart sound by two-thirds of systole. The duration of outward thrust is a reflection of the duration of left ventricular ejection. The passive diastolic filling of the ventricle is not appreciable over the chest wall, though it is at this time that the whole ventricular cavity is expanding to fill itself. Only when the ventricle fills abnormally, a diastolic event is palpable. The right ventricular events underlying the parasternal area are not palpable unless the right ventricle is enlarged or the chest wall is very thin.

The apical impulse is usually in the 5th left intercostal space inside or at the mid-clavicular line. It may be in the 4th intercostal space in children. In thin and tall individuals, the impulse may be more distal (6th space) and medial. In obese people with elevated diaphragm, the impulse may be displaced laterally. Normally it is within 10 cm of the mid-sternal line or 8 cm from the left sternal edge. The normal impulse does not exceed 2.0–2.5 cm in diameter and is never felt in more than one intercostal space. The amplitude of excursion of the impulse is greater in thin persons as a normal variant. The normal impulse is only a gentle tap but no objective measurements are available. Clearly exaggerated amplitude of excursion is easily made out. The amplitude of excursion of the impulse is exaggerated in volume loaded and hyperkinetic circulatory states but the duration of the impulse

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Table 15.1: Normal cardiac impulse

<i>Feature</i>	<i>Description</i>
Site of impulse	Fifth space, at or inside mid clavicular line, 10 cm or less from mid-sternal line
Size of impulse	Less than one and half rib spaces or not more than 2–3 cm
Amplitude of excursion	No objective measurement available Comparison to the experience of normal
Duration of impulse	Less than 50% of systole
Palpable sounds and murmurs	No sound or murmur is palpable over a normal apical impulse
Retraction of apical impulse	The events at apex may not represent left ventricle

is normal. In the majority of adults, the impulse is difficult to palpate. Therefore, a clearly palpable cardiac impulse in a thick chested adult generally indicates cardiac enlargement.

RECORDING THE PRECORDIAL IMPULSE

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The normal apexcardiogram consists of a broad systolic upstroke (Fig. 15.1). The rapid filling wave (rfw) corresponds to the third heart sound. There is a small *a* wave corresponding to atrial contraction. In situations where fourth heart sound is heard, the height of the *a* wave increases. Point *o* corresponds to opening of the mitral valve.

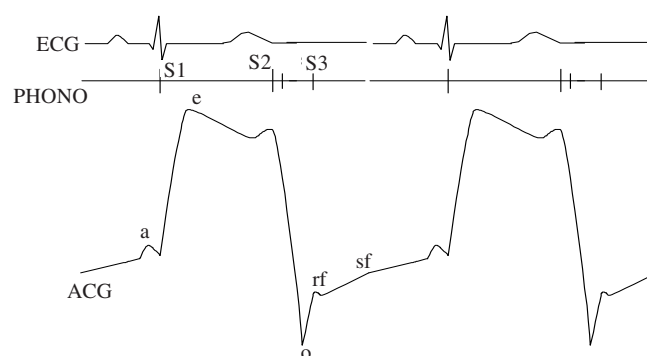


Fig. 15.1: Normal apexcardiogram

S1: first heart sound, S2: second heart sound, S3: third heart sound, ACG: apexcardiogram, ECG: electrocardiogram, Phono: phonocardiogram, a: atrial contraction, o: opening of mitral valve, rf: rapid filling wave, sf: slow filling wave.

Table 15.2: Information obtainable from precordial palpation

<i>Information</i>	<i>Observation</i>
<i>Ventricular enlargement</i>	RV or LV enlargement (lateralization of heart lesion)
If ventricle is enlarged,	Volume load or Pressure load
Abnormalities of systolic emptying	Sustained impulse (pressure load) Systolic thrill across ventricular outflow
Abnormalities of diastolic filling of the ventricles	Palpable third sound Palpable fourth sound Diastolic thrill at apex/tricuspid area
<i>Ventricular dysfunction</i>	Dilatation Sustained impulse
State of great vessels	Palpable vessel + palpable sound (dilatation + high pressure) Impalpable vessel + thrill (Outflow obstruction)
Mediastinal shift due to lung disease	Displacement of apical impulse
Dextrocardia	To determine which side the heart is located

Left ventricular enlargement could be due to

- Volume load
- Pressure load
- Combined volume and pressure load
- Coronary artery disease with
Myocardial infarction
Ventricular aneurysm
- Cardiomyopathy or Myocarditis

Hyperkinetic apical impulse (or forcible impulse)

In volume overload states due to increase in preload and decrease in afterload the extent of myocardial fibre shortening is increased. The apical impulse is then exaggerated in its amplitude of excursion. This type of impulse is characteristic of volume overload states like aortic regurgitation, mitral regurgitation and all other hyperkinetic states (Fig. 15.2). The duration of the impulse is the same as that of the normal impulse. There is no objective method of measuring the amplitude of excursion of the impulse.

THE CARDIAC IMPULSE

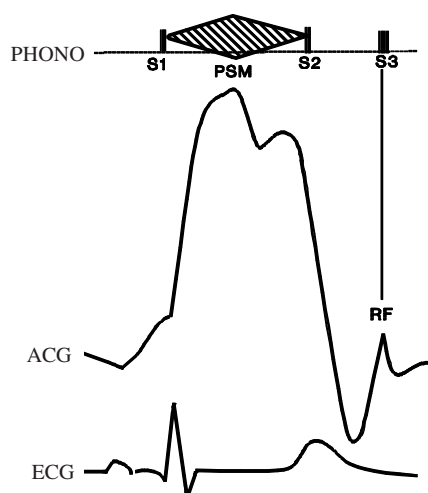


Fig. 15.2: Hyperdynamic apical impulse with rapid filling wave in MR

Sustained apical impulse

When the impulse stays on as an outward movement after the 50 per cent of systole or nearer to the second heart sound, it is called a sustained impulse. The duration of the impulse is a reflection of the duration of the left ventricular ejection (Fig. 15.3).

It is best appreciated by simultaneous auscultation and palpation. Pressure overload of the left ventricle as in severe aortic stenosis or longstanding severe systemic hypertension are usually responsible for a sustained impulse.

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Table 15.4: Causes of volume overload to the left ventricle

<i>Mechanism</i>	<i>Disease</i>
Valvular regurgitations	Mitral regurgitation Aortic regurgitation
Left to right shunts (post-tricuspid)	Ventricular septal defect Patent ductus arteriosus Systemic to pulmonary artery shunt (Blalock-Waterston) Systemic arteriovenous fistula
Hyperkinetic circulatory states	Anemia Thyrotoxicosis Pregnancy Beri-beri

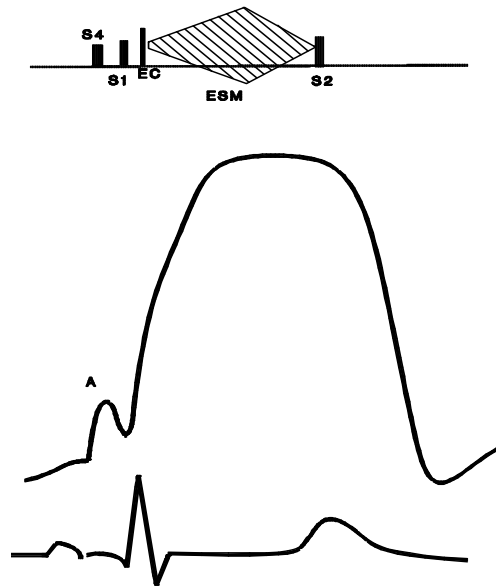


Fig. 15.3: Sustained apical impulse in aortic stenosis

Severe volume overload, severe left ventricular dysfunction due to any condition or a dyskinetic impulse as in coronary artery disease, may also produce a sustained impulse. With severe left ventricular enlargement the normal elliptical left ventricle becomes spherical. This may result in a sustained impulse even up to the second heart sound.

Systolic retraction of apical impulse

As the apex is formed by the interventricular septum adjacent to the left ventricle, the motion of the interventricular septum naturally influences the apical impulse. The normal interventricular septum behaves as if it is a part of the left ventricle and the normal outward apical impulse in systole occurs. In conditions with right ventricular volume load as in atrial septal defect, tricuspid regurgitation or pulmonary regurgitation the interventricular septum behaves as if it is a part of the right ventricle and this anterior motion of the interventricular septum is called *paradoxical septal motion* in the echocardiogram. When the interventricular septum moves along with the right ventricle anteriorly, the apex retracts in systole and moves out in diastole. This apical retraction is the palpable equivalent of paradoxical septal motion in the echocardiogram.

THE CARDIAC IMPULSE

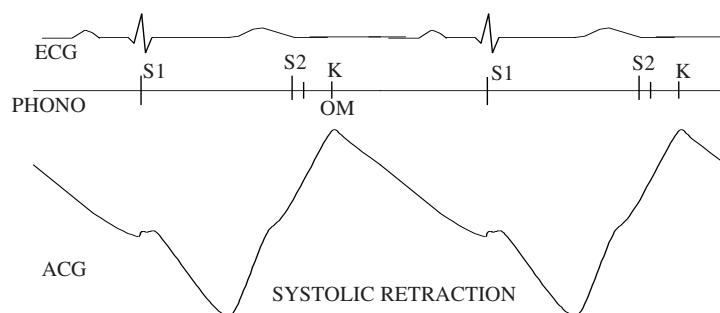


Fig. 15.4: Apexcardiogram of constrictive pericarditis
 S1: first heart sound, S2: second heart sound, K: pericardial knock,
 OM: opening of mitral valve.

The most prominent event in constrictive pericarditis is the rapid ventricular filling. This is appreciated all over the precordium, including the apex. It coincides with the pericardial knock on auscultation and the rapid γ descent in the jugular venous pulse (Fig. 15.4).

On simultaneous auscultation and palpation, the impulse comes out following the second heart sound and moves in following the first heart sound. Unless carefully looked for, this is often mistaken for systolic impulse and some other diagnosis is considered. This most prominent impulse is seen and felt over the left parasternal area.

In the presence of apical retraction any significant left ventricular enlargement is unlikely.

Location of the apical impulse in left ventricular enlargement

In concentric hypertrophy of pressure-loaded states the impulse is not displaced significantly and may stay the same place, that is, the 5th space. In moderate to severe volume-loaded states, the impulse is always displaced downwards and outwards. Apical displacement may be due to non-cardiac disease. In such a case the impulse is neither hyperkinetic nor sustained. An enlarged left ventricle produces a larger impulse that is more than 2 cm or more than one rib space. But if the chest wall is thick or deformed or with obstructive lung disease the impulse may not be palpable in spite of enlargement of the left ventricle. In other words, in these situations even if the impulse is just palpable it may indicate enlargement. A very large left ventricle may in rare cases, produce a left parasternal impulse.

Localized versus diffuse apical impulse

The term diffuse apical impulse is applicable to the situation when severe right ventricular enlargement results in the right ventricle forming the apex with the point of maximal impulse at the parasternal area. The apical impulse in this situation is diffuse and non-localizable. The severe left ventricular enlargement in chronic severe AR or MR or heart failure produce a larger apical impulse but is still localized by definition. This impulse should not be termed diffuse.

Table 15.5: Major types and causes of apical impulse

<i>Type of impulse</i>	<i>Cause</i>
Tapping impulse	Normal impulse with loud S1 Mitral stenosis Atrial septal defect
Hyperkinetic or forcible impulse	Hyperkinetic states Anemia Thyrotoxicosis Pregnancy Regurgitant lesions Mitral regurgitation Aortic regurgitation Left to right shunts Ventricular septal defect Patent ductus arteriosus
Sustained impulse	Pressure loads Systemic hypertension Aortic stenosis Severe volume loads Severe aortic regurgitation Severe MR with left atrial lift Coronary artery disease LV aneurysm Apical infarct or aneurysm Severe LV dysfunction Severe LV dysfunction due to any cause with low ejection fraction Cardiomyopathy (dilated) Myocarditis
Systolic retraction	Constrictive pericarditis RV volume load Atrial septal defect Tricuspid regurgitation Pulmonary regurgitation

Left ventricular wall motion abnormalities in coronary artery disease

Normally except the region of the apex, all the other walls of the left ventricle move inwards to generate pressure inside the chamber. When any of these regions is ischemic or infarcted or thinned and fibrotic, a systolic outward lift may occur in the region between the apex and the left parasternal area (ectopic area). If the region of the apex is involved, a larger and longer impulse may occur simulating a pressure overload. This palpable dyskinesia may occur during ischemia or infarction. If in a patient with chest pain, the impulse appears along with pain but disappears after the subsidence of pain it is diagnostic of myocardial ischemia which must be causing the chest pain. When recognized, its clinical significance cannot be overemphasized. Palpable dyskinetic segment over precordium during angina has more diagnostic value than an exercise test or coronary angiogram, as a coronary angiogram can only reveal an anatomical obstruction but fails to predict whether the patient's chest pain is related to the lesion at hand. In adults (over 40 years), the cardiac impulse is generally difficult to palpate. In these patients, any such palpable impulse should not be ignored.

Palpable diastolic events at the apex

Palpable third heart sound: Normally the diastolic filling of the ventricle during the rapid filling phase and pre-systole are not visible or palpable. When the rapid filling of the ventricle is exaggerated, it results in an abrupt and prominent diastolic wave following the second heart sound and can be seen and palpated. It is normally appreciable in childhood and pregnancy but in adults this generally indicates either a volume-loaded state (mitral regurgitation/aortic regurgitation), hyperkinetic state (anemia) or ventricular failure (Table 15.6). The most prominent rapid filling wave

Table 15.6: Palpable rapid filling wave (third heart sound)

<i>Apex (left ventricle)</i>	<i>Parasternal area (right ventricle)</i>
<i>Physiological</i> Children Pregnancy <i>Pathological</i> Left ventricular failure Mitral regurgitation <i>Hyperkinetic states</i> Anemia Thyrotoxicosis	Right ventricular failure Severe tricuspid regurgitation Pericardial knock

Table 15.7: Palpable *a* wave (pre-systolic impulse)

<i>Apex (left ventricle)</i>	<i>Parasternal area (right ventricle)</i>
Hypertrophic cardiomyopathy	Severe pulmonary hypertension of any cause
Aortic stenosis	Pulmonic stenosis (severe)
Coronary artery disease, acute or chronic	Cardiomyopathy
Acute aortic regurgitation	Right ventricular infarction
Acute mitral regurgitation	Acute tricuspid regurgitation

(third heart sound) is appreciated in severe mitral regurgitation and constrictive pericarditis. In constrictive pericarditis the only outward impulse may be this rapid filling wave; it coincides with the pericardial knock or the third heart sound with systolic retraction of the whole precordium.

Pre-systolic impulse

The pre-systolic atrial contraction distending the ventricle is never palpable in health but may become palpable when the ventricle is non-compliant due to hypertrophy or ischemia (Table 15.7). It is appreciated as an initial hesitant movement of the apex before the bold, sweeping systolic impulse. The anatomical correlate of this is a ventricle with severe hypertrophy and a small cavity (concentric hypertrophy). The hemodynamic correlate of a palpable fourth heart sound is an LV end-diastolic pressure of 15–18 mmHg (normal less than 12). Severe concentric hypertrophy of the left ventricle occurs in severe aortic stenosis, severe systemic hypertension and hypertrophic cardiomyopathy. In coronary artery disease either during ischemia or an acute phase of infarction, a palpable fourth heart sound can occur due to diastolic dysfunction leading to a non-compliant ventricle. In angina pectoris this may appear with pain and may disappear along with subsidence of pain. When the apical impulse is palpable, the fourth heart sound is usually palpable as in aortic stenosis, hypertension and hypertrophic cardiomyopathy. However, when the apical impulse is not palpable, as in the majority of patients with acute myocardial infarction, the fourth heart sound is often not palpable, though heard. The belief that the fourth heart sound is better palpated than heard, is based upon the low frequency components of the sound, but this is applicable only when the left ventricular impulse is available for palpation, as in aortic stenosis, hypertrophic cardiomyopathy or hypertension. When the impulse is not palpable as in coronary artery disease, the fourth heart sound is better heard than palpable. The most

prominent palpable fourth heart sound occurs with hypertrophic cardiomyopathy. Whenever the fourth heart sound or third heart sound are palpable, an AV valve stenosis on that side of the heart is unlikely, as neither the pre-systolic impulse nor the rapid filling of the ventricle are possible with mitral or tricuspid stenosis.

Prolonged diastolic vibrations at apex (left ventricular inflow)

This is suggestive of mitral stenosis and reflects the turbulence to flow during ventricular filling. It is also known as a *diastolic thrill*. As this thrill is localized to the apex, enough care should be taken to locate the apex and graduated pressure should be used to appreciate the thrill. In mitral stenosis a palpable diastolic thrill generally means a mobile non-calcific valve. When the right ventricle is enlarged due to severe pulmonary arterial hypertension and comes to occupy the apex, the diastolic thrill may not be appreciable. The diastolic thrill at the apex may occur in severe rheumatic mitral regurgitation due to the mid-diastolic murmur associated with it, as there is always some degree of anatomical narrowing of the valve in rheumatic heart disease. The associated third heart sound differentiates this from mitral stenosis, as a third heart sound is unlikely in the presence of mitral stenosis. In non-rheumatic mitral regurgitation a diastolic thrill does not occur.

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Left ventricular outflow palpation

A systolic impulse in the aortic area is never normal and indicates a dilated aorta or ascending aortic aneurysm. The systolic thrill indicates aortic stenosis. A systolic thrill only in the carotids but no thrill over the precordium, is suggestive of supra-aortic stenosis.

Right ventricular enlargement

The right ventricle lies under the left lower sternum and any enlargement in the ventricle is most evident there. The normal right ventricle is not palpable except in very thin persons or children, where a brief outward movement followed by retraction is a feature. Right ventricular enlargement is maximal at the 4th or 5th interspace at the left sternal border and directly beneath the lower sternum.

Hyperkinetic left parasternal impulse

This occurs in pure volume overload conditions like atrial septal defect or organic tricuspid regurgitation with normal right ventricular pressures. The amplitude of the lift is increased but not the duration.

Sustained parasternal lift

This occurs in pressure overload states as in pulmonic stenosis or pulmonary arterial hypertension.

Combined hyperkinetic and sustained lift

This can occur in atrial septal defect with pulmonary arterial hypertension, or pulmonary arterial hypertension with tricuspid or pulmonary regurgitation.

Grading of parasternal lift: The parasternal lift is often graded according to the amplitude of lift. This grading does not take the duration of the lift into consideration.

Grade 1: Mild lift made out after careful observation or looking at the chest from a tangential view. May require to use a pencil, or scale to be kept along the parasternal region. May be normal

Grade 2: An obvious lift easily made out

Grade 3: A very prominent lift

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The presence of a parasternal bulge should be commented upon along with the grading. As the grading takes into account only the amplitude of excursion, the duration of the lift should be commented upon additionally (for example, left parasternal lift Grade 2/3, not sustained).

Left parasternal lift of mitral regurgitation (or left atrial lift)

In severe mitral regurgitation, there is a left parasternal lift; it does not indicate right ventricular enlargement. The systolic rise in pressure in the left atrium, produces an expansile impulse (large *v* wave). Part of the left ventricular pressure is transmitted into the left atrium expanding it. The left atrium is bound by the spine posteriorly and any posterior expansion is limited. This results in anterior expansion and displacement of the anterior structures towards the chest wall (the right ventricle, right atrium, pulmonary artery), producing the parasternal lift. This lift is late systolic as it takes time for the left ventricle to fill the left atrium and expand it. It is also more diffuse than the right ventricular impulse. The late onset, diffuseness of the impulse and absence of signs of pulmonary arterial hypertension distinguish this lift from that of right ventricular enlargement. This left atrial lift indicates severe mitral regurgitation with a non-compliant, less dilated left atrium.

Diastolic events over right ventricle

The third heart sound and fourth heart sound may be palpable over the parasternal region and they increase on inspiration and decrease during expiration. The jugular venous pulse counterpart of the third heart sound is rapid γ descent, and that of the fourth heart sound is a prominent a wave.

Palpation of right ventricular outflow (infundibulum of right ventricle and main pulmonary artery): The normal pulmonary artery may be palpable in thin chested persons. A palpable pulmonary artery and pulmonary component of the second heart sound generally indicates pulmonary arterial hypertension. A palpable pulmonary artery with no palpable second sound (pulmonary component) may mean large flow into pulmonary artery without rise in pressures as in left to right shunts. A systolic thrill in the pulmonary area with no palpable pulmonary artery or pulmonary sound associated with a sustained parasternal lift, indicates pulmonic stenosis.

Right ventricular inflow: The diastolic thrill at the left 4th or 5th space, increasing on inspiration indicates tricuspid stenosis. The jugular venous pulse should be viewed as part of the right ventricular inflow as it gives valuable clues to the filling patterns of the right ventricle (γ descent/and a wave). A rapid γ descent correlates with palpable third heart sound, and a slow γ descent with the diastolic thrill of tricuspid stenosis. A raised jugular venous pressure, no γ descent and no palpable third heart sound indicates cardiac tamponade. The prominent a wave in the jugular venous pulse correlates with a palpable fourth heart sound.

Right sternoclavicular joint pulsations

Pulsations of the sternoclavicular joint may be appreciated in a right sided aortic arch, as in tetralogy of Fallot or acute aortic dissection. In tetralogy of Fallot, the Blalock-Taussig anastomosis is performed on the side opposite the aortic arch, while a modified shunt is performed on the same side as the aortic arch. A right aortic arch is a contraindication to the descending aorta to the left pulmonary artery shunt since the bronchus is interposed between the pulmonary artery and the aorta.

TECHNIQUE OF PRECORDIAL EXAMINATION

The patient should be supine or at 45 degrees elevation. Each of us must identify the part of our hand that is most sensitive for detecting thrills or palpable sounds. Once the apical impulse is located, graduated pressure of the hand or fingers is helpful in appreciating a thrill or palpable sound. High frequency events such as first heart sound and second heart sound are best felt by firm pressure. Low frequency events such as third heart sound and fourth heart sound are best felt by light palpation. The patient can be asked to hold the breath in expiration when the heart moves closer to the chest wall. The left lateral position can be used to check the presence or absence of an apical impulse, for palpable thrills or sounds. To assess the site, size, amplitude and duration, the supine position is the best.

The clinical features may alter in people with chest wall deformities.

Causes of non-palpable apical impulse

- Obesity
- Muscular chest wall
- Barrel chest
- Emphysema
- Coronary artery disease with decreased apical motion
- Pleural or pericardial effusion
- Age above 40
- Heart is in a different site (?dextrocardia)

CHARACTERISTICS OF PRECORDIAL MOTION IN VARIOUS CARDIAC ABNORMALITIES

The cardiac impulse in various disorders gives valuable clues to the underlying lesion, its severity, associated lesions and complications.

Cardiac impulse in mitral stenosis

The factors influencing the cardiac impulse in mitral stenosis are:

- Severity of mitral stenosis
- Presence and severity of pulmonary arterial hypertension
- Right ventricular failure and functional TR

Associated factors

- Mitral regurgitation
- Aortic valve disease

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- Tricuspid valve disease
- Atrial septal defect
- Systemic hypertension

Complications

- Calcification of mitral valve
- Rhythm

The typical impulse in mitral stenosis is related to the right ventricular hypertrophy secondary to pulmonary arterial hypertension and the lack of left ventricular enlargement. Classic features of the cardiac impulse in mitral stenosis are:

- The apical impulse is diffuse and is formed by the right ventricle
- Diastolic thrill at apex
- Sustained left parasternal impulse
- Palpable pulmonary arterial pulsations
- Palpable pulmonary sound

Any alteration in the above features or absence of expected features are of importance in mitral stenosis.

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As mentioned in Table 15.8, the most prominent of parasternal impulses is seen when mitral stenosis is complicated by pulmonary arterial hypertension and tricuspid regurgitation, or is associated with atrial septal defect (Lutembacher syndrome).

Mitral regurgitation

The impulse is influenced by the cause, severity, complications and associated lesions in mitral regurgitation. Classic features of the cardiac impulse in this condition are:

- The apical impulse is localized, displaced downwards and outwards
- Hyperkinetic character
- Palpable third heart sound at apex
- Systolic thrill at apex (uncommon)
- Late systolic, diffuse left parasternal left atrial lift
- Palpable pulmonary arterial pulsations and pulmonary sound with pulmonary arterial hypertension

Any deviation from the above features is of importance in the setting of mitral regurgitation.

Table 15.8: Cardiac impulse in mitral stenosis

<i>Feature</i>	<i>Alterations/significance</i>
Apical impulse Site	Normal with mild mitral stenosis Lateral displacement in moderate to severe mitral stenosis
Diastolic thrill	Is common with moderate to severe mitral stenosis Absent with Extremes of obstruction Mild obstruction with little or no turbulence Severe MS with severe PAH, RVF and TR with low cardiac output Indicates a relatively mobile mitral valve, severe calcification is unlikely
Diffuse impulse	RV forming the apex in moderate to severe mitral stenosis
Localized impulse	Mild mitral stenosis Associated Aortic valve disease Mitral regurgitation Anemia Systemic hypertension Thyrotoxicosis Associated CAD Rheumatic myocarditis
Left parasternal impulse (LPSI) Normal	Mild mitral stenosis Moderate mitral stenosis and PAH in adults with thick chest wall Associated tricuspid stenosis
Sustained parasternal impulse (> Gr 2/3)	Indicates moderate or severe mitral stenosis with PAH
Sustained + hyperkinetic LPSI (Gr 3/3)	Mitral stenosis + PAH Tricuspid regurgitation Pulmonary regurgitation Associated atrial septal defect
Palpable pre-systolic impulse (RV S4) along left sternal border	Severe PAH with concentric RVH, non-compliant RV Correlates with pronounced <i>a</i> wave in JVP Rules out associated tricuspid stenosis
Palpable rapid filling wave (RV S3) along LSB	Right ventricular failure Correlates with rapid <i>y</i> descent
Palpable P2/Pulmonary artery Systolic thrill	Pulmonary arterial hypertension Associated atrial septal defect (Lutembacher syndrome)

Table 15.9: Significance of cardiac impulse in mitral regurgitation

<i>Feature</i>	<i>Significance</i>
Apical impulse	
<i>Site</i>	
Normal	Mild mitral regurgitation AS mistaken for mitral regurgitation
Downward displacement	Moderate to severe mitral regurgitation
<i>Character</i>	
Hyperkinetic	Moderate to severe mitral regurgitation AS mistaken for mitral regurgitation
Sustained impulse	Mitral regurgitation secondary to coronary artery disease HOCM Congestive cardiomyopathy Severe mitral regurgitation with LV dysfunction
<i>Systolic thrill</i>	Less common May occur with moderate to severe mitral regurgitation Chordal rupture Rule out AS or VSD simulating mitral regurgitation
<i>Diastolic thrill</i>	Associated mitral stenosis Indicates severe rheumatic mitral regurgitation in the absence of mitral stenosis
<i>Palpable S3</i>	Mitral regurgitation is more likely than AS Indicates moderate mitral regurgitation in adults Rules out associated mitral stenosis
<i>Palpable S4</i>	AS may have been mistaken for MR Rules out rheumatic mitral regurgitation Mitral regurgitation in association with HOCM Restrictive cardiomyopathy Coronary artery disease
<i>Late, diffuse parasternal lift</i>	Left atrial lift with severe mitral regurgitation
<i>Early LLSB lift</i>	RVH due to pulmonary arterial hypertension

In acute mitral regurgitation, the murmur of MR may be faint or absent and the only clue to the underlying MR is unexplained hyperkinesia of the apical impulse.

Cardiac impulse in aortic stenosis

The cardiac impulse in aortic stenosis depends on the severity, nature, associated lesions and complications of aortic stenosis. The classic cardiac impulse in aortic stenosis is located in the 5th space without displacement, and is of sustained duration

Table 15.10: Cardiac impulse in aortic stenosis

<i>Feature</i>	<i>Significance</i>
Normal	Mild aortic stenosis Aortic valve sclerosis May occur even with severe aortic stenosis
Displaced downward and outward	Mitral regurgitation mistaken for aortic stenosis AS complicated by CCF Associated Aortic regurgitation patent ductus arteriosus ventricular septal defect Myocardial infarction
Sustained impulse	Moderate to severe aortic stenosis Mild aortic stenosis with associated Systemic hypertension Severe aortic regurgitation Large patent ductus arteriosus Myocardial infarction
Systolic thrill at Aortic area (Right 2 nd space) Carotids (Right)	Favours organic AS in the presence of severe AR Can occur in functional AS with severe aortic regurgitation
Left sternal border	May favour sub-valvular obstruction
Apex	May simulate mitral regurgitation May be the only site of thrill in calcific AS in elderly with severe emphysema
Carotids only	Supravalvular aortic stenosis Carotid stenosis Takayasu's arteritis
Palpable S4	Favours aortic stenosis over mitral regurgitation Absence of this sign makes HOCM unlikely Indicates severe obstruction
Palpable S3	Rules out associated mitral stenosis Mitral regurgitation mistaken for aortic stenosis Aortic stenosis complicated by LVF Associated mitral regurgitation, PDA or VSD

(Fig. 15.3); the fourth heart sound may be palpable. A systolic thrill is felt over the left sternal border, aortic area and carotid artery.

Any deviation from this classic impulse may mean either an associated lesion or a complication of aortic stenosis. In the majority of patients even with severe

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aortic stenosis, the cardiac impulse is normally located due to concentric left ventricular hypertrophy without much dilatation of the cavity. Once the impulse is significantly displaced, an associated disorder enumerated above should be considered.

Cardiac impulse in aortic regurgitation

The determinants of the nature of the impulse are the severity and the presence of associated disorders. The classic features are:

- The impulse is displaced outward and downward

Table 15.11: Significance of the cardiac impulse in aortic regurgitation

<i>Feature</i>	<i>Significance</i>
<i>Location</i>	
Normal	Mild aortic regurgitation
Displaced downward and outward	Moderate to severe aortic regurgitation Mild aortic regurgitation with associated mitral regurgitation ventricular septal defect coarctation patent ductus arteriosus
Hyperkinetic impulse	Suggests moderate to severe aortic regurgitation Mild aortic regurgitation with associated mitral regurgitation ventricular septal defect patent ductus arteriosus
Palpable S3	Suggestive of Left ventricular failure Associated mitral regurgitation
Palpable S4	Acute aortic regurgitation Associated hypertension Aortic regurgitation with acute aortic dissection
Diastolic thrill	Rare Suggests retroversion of aortic cusps as in aortic root disease
<i>Systolic thrill</i>	Is often mistaken for systolic thrill
Right 2 nd space	Associated aortic stenosis
Right 2 nd space and carotids	Functional aortic stenosis in severe aortic regurgitation
Only carotids	Aortic regurgitation in association with Takayasu's arteritis
Left sternal border only	Subaortic obstruction Associated ventricular septal defect

- Hyperkinetic or forcible in nature
- Pulsations of ascending aorta may be palpable at right 2nd space
- Systolic thrill may be palpable over the carotids
- No additional sounds are palpable

Any alteration from the above or any additional sign is of significance in the setting of aortic regurgitation (Table 15.11).

In pure severe aortic regurgitation, a systolic thrill over the carotids is common and does not indicate associated aortic stenosis. The systolic thrill along the left sternal border should be carefully interpreted in the setting of aortic regurgitation. The systolic thrill along the left sternal border with accompanying thrill at right 2nd space and carotid is suggestive of valvular aortic stenosis. A similar thrill at the lower sternal border without associated thrill at right 2nd space and carotid may indicate either subaortic stenosis or ventricular septal defect.

Cardiac impulse in congenital acyanotic heart disease

Evaluation of the precordial impulse is of great value in the assessment of this group of disorders.

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Pulmonic stenosis

The classic features are:

- Apical impulse is diffuse
- Left parasternal impulse Grade 2/3
- Sustained
- Systolic thrill at pulmonary area
- Impalpable pulmonary artery
- Palpable pre-systolic impulse at LSB

Any deviation from these classic features is of significance in the recognition of pulmonary stenosis. Very often, even in severe pulmonic stenosis, the left parasternal impulse is unimpressive as it is a pure pressure load to the right ventricle (Table 15.12).

Atrial septal defect

The atrial septal defect is the purest example of volume load to the right ventricle.

The classic features are:

- The apical impulse is diffuse formed by RV with systolic retraction

THE CARDIAC IMPULSE

Table 15.12: Significance of cardiac impulse in pulmonic stenosis

<i>Feature</i>	<i>Significance</i>
<i>Apical impulse</i>	
Diffuse	RV may form the apex
Localized	Ventricular septal defect or AS mistaken for PS As a variant in pulmonic stenosis(rare)
Systolic retraction	Atrial septal defect mistaken for pulmonic stenosis Associated TR
<i>Left parasternal impulse</i>	
Grade 1–2/3	Consistent with pulmonic stenosis
Grade 3/3	Unlikely to be pure pulmonic stenosis Associated atrial septal defect or TR likely
Sustained impulse	Moderate to severe pulmonic stenosis
<i>Palpable S4</i>	Indicates severe pulmonic stenosis Favours pulmonic stenosis with intact ventricular septum
<i>Palpable S3</i>	Favours intact atrial septum Associated RV failure
<i>Systolic thrill</i>	
Pulmonary area	Valvular pulmonic stenosis
Left 2 nd space	Infundibular pulmonic stenosis
Left 3 rd or 4 th space	Infundibular pulmonic stenosis
Infraclavicular/laterally to pulmonary area	Supravalvular pulmonic stenosis
<i>Diastolic thrill</i>	Associated pulmonary incompetence as in Dysplastic valve of Noonan's syndrome Infective endocarditis Absent pulmonary valve with annular stenosis

- Hyperkinetic left parasternal impulse Grade 2/3
- Palpable pulmonary arterial pulsations at left 2nd space
- P2 may be palpable
- Systolic thrill at pulmonary area in 25 per cent of cases

Any variation from the above features may signify an associated disorder or a complicating feature (Table 15.13).

Ventricular septal defect

In ventricular septal defect, the left ventricle is volume loaded and the right ventricle

Table 15.13: Significance of cardiac impulse in atrial septal defect

<i>Feature</i>	<i>Significance</i>
<i>Apical impulse</i>	
Diffuse	RV enlargement forming apex
Localized	LV enlargement due to associated mitral regurgitation as in MVP/RHD or AV canal defects VSD mistaken for atrial septal defect or associated VSD
Systolic retraction	RV volume load
<i>Left parasternal impulse</i>	Suggests left to right shunt > 1.5:1
Hyperkinetic 2/3	Consistent with RV volume load due to atrial septal defect
Sustained impulse	Atrial septal defect with pulmonary arterial hypertension Atrial septal defect with pulmonic stenosis Atrial septal defect with mitral valve disease
<i>Palpable S4</i>	Primary PAH simulating atrial septal defect is likely
<i>Palpable S3</i>	RV failure
<i>Diastolic thrill</i>	Ebstein's anomaly is more likely than atrial septal defect
<i>Pulmonary area</i>	
Pulsations of PA	Favours atrial septal defect over pulmonic stenosis
Palpable P2	May occur due to large shunt or pulmonary arterial hypertension
<i>Systolic thrill</i>	Various possibilities exist Pulmonic stenosis is more likely than atrial septal defect Associated pulmonic stenosis with atrial septal defect Associated mitral valve disease (Lutembacher syndrome) Ostium primum atrial septal defect Some of atrial septal defects, 25% may have this sign without associated disease; suggests large shunt

is both volume-and pressure-loaded.

The classic features are:

- Apical impulse localized and hyperkinetic
- Left parasternal impulse is hyperkinetic and sustained
- Systolic thrill anywhere along the left sternal border
- Pulsations of PA at pulmonary area
- P2 may be palpable

THE CARDIAC IMPULSE

Each of these features can be modified by the size, location, associated defects and complications of ventricular septal defect.

Other determinants are

- Site of defect
- Location of the defect
- Complications: pulmonary arterial hypertension
- Congestive cardiac failure

Table 15.14: Significance of cardiac impulse in ventricular septal defect

<i>Feature</i>	<i>Significance</i>
<i>Apical impulse</i>	
Normal	Small ventricular septal defect
Localized, Hyperkinetic	Moderate to large ventricular septal defect Continuing left to right shunt
Sustained impulse	AS mistaken for ventricular septal defect Associated coarctation Associated AS
Diffuse	RV forming apex as in atrial septal defect
<i>Left parasternal impulse</i>	
Hyperkinetic	Moderate ventricular septal defect without PAH
Hyperkinetic and sustained	Moderate or large defect with PAH
Combined apical + left parasternal impulse	Ventricular septal defect with left to right shunt
Isolated left parasternal impulse	VSD with PAH with little or no left to right shunt
Isolated hyperkinetic apex	MR mistaken for ventricular septal defect Small VSD with significant aortic regurgitation
Palpable pulmonary arterial pulsations	Favours VSD over pulmonic stenosis Significant left to right shunt or PAH
Palpable pulmonary sound	Suggests PAH and rules out pulmonic stenosis
<i>Systolic thrill</i>	
Left sternal border	Consistent with ventricular septal defect
Pulmonary area	Supracristal ventricular septal defect
Right sternal border	LV to RA communication (Gerbode's defect)
Palpable third heart sound	Heart failure Associated mitral regurgitation
Palpable fourth heart sound	VSD is unlikely; pulmonic stenosis is more likely PAH with TR is also likely

Table 15.15: Significance of cardiac impulse in patent ductus arteriosus

<i>Feature</i>	<i>Significance</i>
<i>Apical impulse</i>	
Normal	Small ductus Venous hum mistaken for patent ductus arteriosus
Displaced downward and Hyperkinetic	Moderate to large ductus with significant left to right shunt
Sustained impulse	Very large ductus with large shunt and associated aortic stenosis coarctation of aorta systemic hypertension
<i>Left parasternal impulse</i>	
Normal	No pulmonary arterial hypertension Small ductus
Sustained with hyperkinetic apical impulse	Hyperkinetic pulmonary arterial hypertension due to large pulmonary flow
Sustained with diffuse apical impulse	'Fixed' pulmonary arterial hypertension due to increased pulmonary vascular resistance
Sustained and hyperkinetic with diffuse apical impulse	'Fixed' pulmonary arterial hypertension with pulmonary or tricuspid incompetence or both
<i>Thrill at pulmonary area</i>	
Continuous	Consistent with ductus
Systolic only	Ductus with pulmonary arterial hypertension
Diastolic only	Rules out patent ductus arteriosus with left to right shunt Severe pulmonary arterial hypertension with pulmonary incompetence
<i>Thrill at other sites</i>	
Left sternal border	Ventricular septal defect or aortic stenosis
Aortic area	Aortic stenosis
Suprasternal notch	Consistent with ductus Favours ductus over ventricular septal defect
Palpable pulmonary arterial pulsations and palpable pulmonary sound	Implies moderate to large ductus Pulmonary arterial hypertension

- Associated defects: aortic regurgitation, pulmonic stenosis, patent ductus arteriosus
- Coarctation of aorta

Table 15.14 gives the significance of any deviation from the classic pattern expected in ventricular septal defect.

With isolated parasternal impulse without an accompanying left ventricular enlargement, ventricular septal defect is highly unlikely.

Patent ductus arteriosus

The left ventricle is volume-loaded and the right ventricle is excluded from the shunt in patent ductus arteriosus. The determinants of the impulse are the size of the ductus, the degree of pulmonary arterial hypertension and the presence or absence of associated lesions.

The classic features are:

- Apical impulse is localized and displaced downward and outward
- Hyperkinetic apical impulse
- Continuous or dominantly systolic thrill at pulmonary area
- Pulmonary arterial pulsations may be palpable
- Pulmonary sound may be palpable
- Pulsations over suprasternal notch

Any alteration in any of these features is of significance in the assessment of patients with patent ductus arteriosus (Table 15.15).

A continuous thrill at the pulmonary area with left ventricular enlargement and collapsing pulse is virtually diagnostic of patent ductus arteriosus. The only other condition that produces a continuous thrill at this site is the peripheral pulmonary stenosis. The isolated right ventricular enlargement distinguishes the two conditions.

Cardiac impulse in congenital cyanotic heart disease

Cyanosis in congenital heart disease is due to right to left shunt, abnormal origin of great vessels or admixture of blood in one of the four chambers. Careful evaluation of cardiac impulse gives clues to the underlying mechanism in the majority of patients. The first question one asks in congenital cyanotic heart disease is whether there is a cyanotic heart disease at all in this patient. If present, is it due to diminished pulmonary flow or increased pulmonary flow.

Evaluation

Evaluation of the cardiac impulse gives clues in some of the above situations.

The classic cardiac impulse in some of the common cyanotic heart diseases is described below.

Tetralogy of Fallot

The classic features are:

- Absence of cardiac enlargement
- Apical impulse is normal or unimpressive
- Left parasternal impulse < Grade 2/3

Is it CHD or a condition simulating it?	<i>Conditions simulating congenital cyanotic heart disease</i> Methemoglobinemia: congenital or acquired Primary pulmonary hypertension with right to left atrial shunt Chronic cor pulmonale
If it is CHD, is it due to increased pulmonary flow or to diminished pulmonary flow?	<i>Decreased pulmonary flow conditions</i> Tetralogy of Fallot All conditions with tetralogy like physiology DORV with pulmonic stenosis TGA with ventricular septal defect and pulmonic stenosis Single ventricle with pulmonic stenosis <i>Increased pulmonary flow conditions</i> Transposition of great vessels TAPVC DORV Truncus arteriosus Single ventricle

Table 15.16: Evaluation of the cardiac impulse in congenital heart disease

<i>Clues and features</i>	<i>Increased pulmonary flow</i>	<i>Decreased pulmonary flow</i>
Clues from cardiac impulse	Cardiac enlargement Biventricular enlargement Systolic thrill at lower left sternal border (ventricular septal defect) Palpable third heart sound Palpable pulmonary artery Palpable pulmonary sound	Absence of cardiac enlargement Parasternal impulse < Grade 2/3 Systolic thrill at pulmonary area Impalpable pulmonary artery
Accompanying features	Presence of heart failure Elevated jugular venous pressure Presence of third sound Presence of mid-diastolic murmurs at AV valves	Absence of heart failure Normal level of jugular venous pulse Absence of third heart sound Absence of diastolic murmurs at AV valves

THE CARDIAC IMPULSE

- No pulsations in pulmonary area
- Usually no systolic thrill at pulmonary area

The cardiac impulse in tetralogy of Fallot can be almost normal and unimpressive (Table 15.17).

The differential diagnosis of congenital heart disease is best approached by considering ventricular enlargement (Table 15.18). Usually the right ventricle is the enlarged ventricle in congenital heart disease; left ventricular or biventricular enlargement is less common.

Table 15.17: Significance of cardiac impulse in tetralogy of Fallot

<i>Feature</i>	<i>Significance</i>
<i>Apical impulse</i>	
Normal	Consistent with tetralogy of Fallot
Unimpressive or impalpable	As above
Localized, hyperkinetic	Pink tetralogy with left to right shunt or ventricular septal defect Associated aortic regurgitation with tetralogy of Fallot
<i>Left parasternal impulse</i>	
Normal or < Grade 2/3 impulse	Consistent with tetralogy of Fallot
More than Grade 2/3	Tetralogy of Fallot is unlikely Pulmonary stenosis with intact ventricular septum and atrial right to left shunt is likely
<i>Systolic thrill</i>	
With mild cyanosis	Milder tetralogy
With deep cyanosis	Tetralogy is unlikely Conditions with obligatory ventricular septal defect likely tricuspid atresia double outlet right ventricle Pink tetralogy or VSD mistaken for tetralogy of Fallot
With no cyanosis	
<i>Systolic and diastolic thrill at pulmonary area</i>	Pulmonary regurgitation with absent pulmonary valve
<i>Continuous thrill</i>	Associated patent ductus arteriosus
<i>Palpable fourth heart sound</i>	Tetralogy of Fallot is unlikely Pure pulmonic stenosis with right to left atrial shunt is likely
<i>Pulsations at left 2nd space</i>	Though unusual may occur due to outflow pulsations

Table 15.18: Ventricular enlargement in cyanotic heart disease

<i>Conditions with RVH</i>	<i>Conditions with LVH</i>	<i>Conditions with BVH</i>	<i>Conditions with no ventricular enlargement</i>
TOF TGA, VSD + PS DORV with PS TAPVC Trilogy of Fallot	Tricuspid atresia Pulmonary atresia with intact IVS Hypoplastic RV ASD	DORV without PS TGA, VSD without PS Truncus arteriosus Pulmonary atresia with bronchial collaterals or PDA Tetralogy of Fallot with PDA after shunt	Pulmonary AV fistula SVC or IVC draining to LA

Cardiac impulse in coronary artery disease

Most physicians do not care to palpate the precordium when evaluating patients with coronary artery disease (CAD) as the cardiac impulse is often normal in the majority of patients. However, when it is carefully evaluated, it gives valuable clues in a significant number of patients. The determinants of the impulse are the clinical subset, presence or absence of angina during examination, extent of coronary artery involvement, presence or absence of myocardial infarction, the duration of infarction, presence or absence of mitral regurgitation or ventricular septal defect, and the associated disorders like systemic hypertension (Table 15.19).

The area of the precordium over the third, fourth and fifth interspace to the left of the sternum and apex is called the *ectopic area*. In the initial evaluation of patients presenting with angina pectoris, one should rule out conditions producing angina as a secondary manifestation. These are hypertrophic cardiomyopathy, mitral

Table 15.19: Significance of the cardiac impulse in coronary artery disease

<i>Clinical subset</i>	<i>Findings</i>
Chronic stable angina	The impulse is most commonly normal <i>During the episode of angina</i> Palpable fourth heart sound Palpable dyskinesia (ectopic impulse)
Acute myocardial infarction	Palpable fourth heart sound Palpable dyskinesia Systolic thrill at apex in mitral regurgitation due to PMD Systolic thrill at left sternal border in VSD

valve prolapse, severe systemic hypertension, cardiomyopathy and/or severe pulmonary hypertension (thromboembolic or primary).

The majority of patients with coronary artery disease are above 40 years of age and have a thick chest wall making it difficult to palpate the cardiac impulse. When the cardiac impulse is easily palpable in this group of people, cardiac enlargement is most likely. It is for this reason that any easily discernible impulse has diagnostic significance in this age group. Palpable fourth heart sound and dyskinesia appearing during the episode of pain and disappearing later with the subsidence of pain, is virtually diagnostic of ischemic origin of pain.

Cardiac impulse in pericardial disease

a) *Pericardial effusion*: The most common feature in moderate to large pericardial effusions is increase in cardiac dullness with impalpable cardiac impulse. If the cardiac impulse is well felt in the presence of large effusion, associated intracardiac defect with cardiac enlargement is likely. Significant pericardial effusion can co-exist with intracardiac defect in acute rheumatic fever with pancarditis, endomyocardial fibrosis, acute myopericarditis, and uremic pericarditis. In uremic pericarditis due to chronic renal failure, underlying cardiac enlargement due to preexisting systemic hypertension is common.

b) *Chronic constrictive pericarditis*: The cardiac impulse is impalpable in the majority of adults with this disease. In younger patients with thin chest wall the impulse is often palpable and can be prominent. On careful examination the impulse will be found to be retracting in systole with a prominent diastolic outward movement. The impulse reflects the pathophysiology of constrictive pericarditis, namely the very rapid ventricular filling coinciding with pericardial knock and a rapid y descent

Table 15.20: Cardiac impulse in conditions simulating coronary artery disease

Condition	Cardiac impulse
Hypertrophic cardiomyopathy	Left ventricular hypertrophy Palpable fourth sound
Systemic hypertension	As above
Mitral valve prolapse	Systolic thrill of mitral regurgitation Bifid apical impulse Hyperkinetic left ventricular impulse
Severe pulmonary arterial hypertension	Left parasternal impulse, sustained Palpable PA and P2

in the neck veins. This diastolic outward impulse is often mistaken for systolic, and some other diagnosis is considered. It is best detected by simultaneously palpating the parasternal impulse, and auscultating the pulmonary area. The impulse occurs following the second heart sound. Timing with carotid impulse is often misleading particularly with rapid heart rates. It is important to look for this sign because other conditions simulating constriction very rarely produce this sign.

Cardiac impulse in cardiomyopathy

The cardiac impulse in cardiomyopathy gives clues as to the nature of cardiomyopathy (Table 15.21). In congestive cardiomyopathy, systolic dysfunction with exaggeration of rapid ventricular filling is the central feature.

In restrictive cardiomyopathy the systolic function is normal. There is restriction to ventricular filling, calling for exaggerated atrial contraction trying to distend a non-compliant ventricle, resulting in fourth heart sound or pre-systolic impulse. In hypertrophic cardiomyopathy, not only is the restriction to ventricular filling a fundamental problem but a varying degree of obstruction to left ventricular outflow may also occur. As cardiomyopathy is always a diagnosis of exclusion, certain features in the cardiac impulse help to rule out conditions that simulate cardiomyopathy.

Features unusual for a diagnosis of cardiomyopathy are:

- Signs of significant pulmonary arterial hypertension
 - Parasternal impulse > Grade 2/3
 - Palpable pulmonary arterial pulsations
 - Palpable pulmonary sound

Table 15.21: Features of cardiac impulse in cardiomyopathy

<i>Feature</i>	<i>Congestive</i>	<i>Restrictive</i>	<i>Hypertrophic</i>
Apical impulse			
Location	Displaced	Normal	Normal
Diffuse bulge	Common	No	May occur
Palpable S4	No	Common	Common
Palpable S3	Common	May occur	May occur
Systolic thrill	No	No	May occur with LV outflow obstruction

THE CARDIAC IMPULSE

- Systolic or diastolic thrill anywhere over the precordium
- Impalpable third or fourth heart sound even with careful palpation
- Systolic retraction of cardiac impulse

With careful palpation a third heart sound or fourth heart sound can be felt in cardiomyopathies. Absence of this sign makes the diagnosis suspect. As constrictive pericarditis simulates restrictive cardiomyopathy so closely, a systolic retraction of the impulse should be looked for in all patients when either diagnosis is considered. The diagnosis of cardiomyopathy is unlikely when a systolic or diastolic thrill is palpable. The exceptions to this are rare but may occur with hypertrophic obstructive cardiomyopathy (systolic thrill), or endomyocardial fibrosis with severe mitral or tricuspid regurgitation (systolic or diastolic thrill).

In HOCM, there will be a triple impulse due to prominent *a* wave and bifid systolic wave. The notch in systole represents the onset of obstruction (Fig. 15.5).

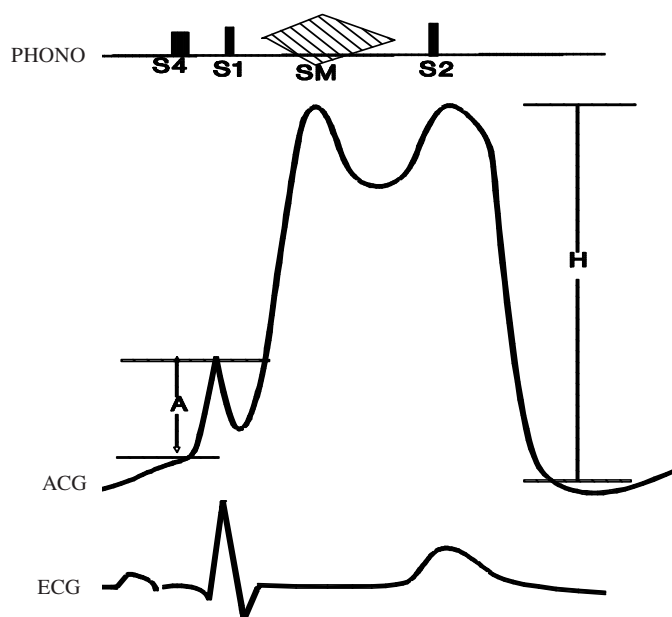


Fig. 15.5: Apexcardiogram in hypertrophic obstructive cardiomyopathy (HOCM)
S1: first heart sound, S2: second heart sound, S4: fourth heart sound, SM: systolic murmur, A: atrial contraction, ACG: apexcardiogram, ECG: electrocardiogram, Phono: phonocardiogram

PRACTICE IMPLICATIONS

- The most basic information you can get out of cardiac impulse is whether the heart is on the right side or left side. Your search is incomplete till you turn the patient to the left side to locate the impulse; if it cannot be found, look for it on the right side. This simple measure could save you the humiliation of missing a case of dextrocardia.
- If the cardiac enlargement (left ventricular or right ventricular) is unexpected, or is out of proportion to the severity of lesion, look for a chest deformity.
- When you make a diagnosis of mitral stenosis, go out of the way to look for the diastolic thrill at apex. If a student fails to detect it but the examiner palpates thrill, the implications are unspeakable.
- In the presence of extreme enlargement of one ventricle, mild enlargement of the other ventricle is difficult to detect.
- One has to identify the ventricular chamber that has hypertrophied.
- Very rarely, even experienced physicians and cardiologists mistake enlargement of one ventricle for the other. I have mistaken myocarditis for primary pulmonary arterial hypertension, hypertrophic cardiomyopathy for right ventricular hypertrophy, and the right ventricular hypertrophy in severe pulmonic stenosis for left ventricular hypertrophy.
- It is often said that the fourth heart sound is better palpable than audible. This however depends whether you are good in palpation or auscultation or both. Palpation is better when the cardiac impulse is palpable. With a loud murmur of aortic stenosis masking the fourth heart sound, palpation is superior to auscultation.
- When the electrocardiogram shows a pattern of LVH, but no LVH is detected on palpation and echocardiogram, apical cardiomyopathy is likely. The transesophageal echocardiography helps to confirm the diagnosis.
- As mistakes are common in the evaluation of cardiac impulse, examiners should try not to be too harsh in judging students. What looks typical and easy may not be so from the point of view of a student facing an examination. (This should be borne in mind by examiners who pretend infallibility.)

16 Auscultation of the Heart

In the naked light, ten thousand people, may be more – people talking without speaking, people hearing without listening..

Paul Simon in *Sound of Silence*

When the stage of auscultation is reached, valuable information has already been obtained from history and physical examination. Though we routinely try to hear any sound or murmur, we often try to check for conditions guided by the information already available. Looking for the mid-diastolic murmur of mitral stenosis in a patient presenting with dyspnea, paroxysmal nocturnal dyspnea and orthopnea is an example. On the other hand, one tries to hear a ventricular gallop or third heart sound if the left ventricular impulse is prominent, for evidence of left ventricular failure. This clinical skill or faculty of ‘looking for things’ on auscultation distinguishes a good clinician from an average one.

For a proper understanding of auscultatory findings, it is important to understand certain commonly used terms.

Frequency is sometimes referred to as pitch. Frequency is an objective expression of the number of vibrations per second; pitch is the subjective description of frequency. Intensity and loudness are the same as intensity is expressed as decibels and loudness is the subjective expression of the same.

High frequency sounds or murmurs

High frequency sounds are usually described as soft, blowing, musical or cooing. High frequency sounds (400 cps) are sharp and clicky as in the two components of second heart sound, ejection clicks and non-ejection clicks. The murmurs of mitral regurgitation, aortic regurgitation and tricuspid regurgitation are often of

Table 16.1: What to 'look' for auscultation

<i>Guiding clue</i>	<i>What to look for</i>
Dyspnea, PND, orthopnoea With no LV enlargement With LV enlargement Chest pain, palpitation in a young patient Febrile patient	Mid-diastolic murmur of MS Third or fourth heart sound Non-ejection click, late systolic murmur of mitral valve prolapse Heart murmur, particularly early diastolic murmur of AR by keeping the patient in sitting, lean-forward position, with held expiration, and diaphragm of stethoscope well pressed to the chest wall soft systolic murmur of MR ejection click of bicuspid aortic valve
Fever with asymmetry of arterial pulses	As above
Fever with or without chest pain	Pericardial rub
Acute chest pain with asymmetry of arterial pulses	Early diastolic murmur of AR complicating aortic dissection
Paradoxical pulse, elevated JVP, slow γ descent, impalpable cardiac impulse	Pericardial rub, absence of third heart sound suggestive of cardiac tamponade

Terms used in the study of sound or auscultation

Acoustics is the science of sound and its effects on people.

Beats are periodic variations in the loudness of sound. Beats are heard when two tones of slightly different frequencies are sounded at the same time.

Decibel is the unit used to measure the intensity of a sound. A 3,000 Hertz tone of about zero decibels is the weakest sound that the normal human ear can hear.

Frequency of sound waves refers to the number of compressions or rarefactions produced by a vibrating object each second.

Pitch is the degree of 'highness' or 'lowness' of a sound as perceived by the listener. It is the subjective expression of frequency.

Hertz is the unit used to measure frequency. One Hertz equals one cycle (vibration) per second.

Intensity of a sound is related to the amount of energy flowing in the sound waves.

Sound quality, also called timber, is a characteristic of musical sounds. Sound quality distinguishes between notes of the same frequency and intensity produced by different musical instruments.

Ultrasound is sound with frequencies above the range of human hearing.

Infrasound is sound with frequencies below the range of human hearing.

AUSCULTATION OF THE HEART

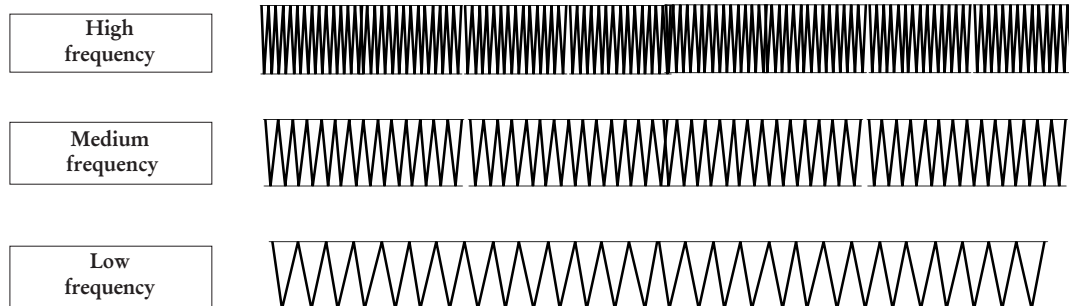


Fig. 16.1: Diagrammatic representation of frequencies

high frequency. The high pitched sounds, or murmurs are best appreciated with the diaphragm of the stethoscope and are widely conducted. Thrills are uncommon with these murmurs. They are often produced when the pressure difference is high.

Low frequency sounds or murmurs

Low frequency sounds are usually described as rough, rumbling, dull and thud. Low frequency sounds (60–200 cps) are dull (for example, the first heart sound, third heart sound and fourth heart sound). The murmurs of mitral stenosis and tricuspid stenosis are low pitched. All low frequency sounds and murmurs are best appreciated with the bell of the stethoscope applied lightly over the chest. Low pitched murmurs and sounds have less number of vibrations per second, but have increased amplitude of vibrations. For this reason they are often palpable (third heart sound/fourth heart sound) or have thrills (mitral stenosis/tricuspid stenosis). Low pitched murmurs usually suggest low pressure difference between the two chambers in the heart (left atrium to left ventricle as in mitral stenosis, or right atrium to right ventricle as in tricuspid stenosis). The murmurs of aortic stenosis, pulmonic stenosis and ventricular septal defect are often described as harsh in quality. A harsh murmur has a combination of frequencies and the low frequency component is localized to the site of best audibility but the high frequency component is widely transmitted. This is most common with the murmur of aortic stenosis where the rough component of the murmur is best heard at the aortic area but the high frequency soft component is transmitted to the apex and is often confused for mitral regurgitation.

Table 16.2: High frequency and low frequency events

<i>Frequency</i>	<i>Description</i>	<i>Examples</i>
High frequency (about 400 cps)	<i>Sounds</i> Sharp Clicky <i>Murmurs</i> Soft Blowing Cooing Musical	<i>Sounds</i> Aortic and pulmonary Ejection clicks Opening snap First heart sound in MS Non-ejection clicks Pericardial knock <i>Murmurs</i> EDM of AR/PR MR Functional TR
Low frequency (60–200 cps)	<i>Sounds</i> Dull Thud <i>Murmurs</i> Rough Rumbling	<i>Sounds</i> Third heart sound Fourth heart sound Pericardial knock <i>Murmurs</i> MDM of MS/TS Austin Flint murmur Flow murmurs at AV valves PR with normal PA pressures
Mixed frequency (combination of high and low frequencies)	<i>Sounds</i> Not as sharp as second heart sound or as dull as third heart sound <i>Murmurs</i> Rough Harsh	<i>Sounds</i> First heart sound <i>Murmurs</i> AS PS VSD

17 The First Heart Sound

The first heart sound is produced by the closure and after vibrations at the mitral and tricuspid valves. The mitral component occurs first followed by the tricuspid component. Additionally, the myocardial and vascular components contribute to it. Heart sounds and murmurs are due to the following mechanisms:

- Valvular
- Myocardial
- Vascular
- Vibration of the cardiohemic system

Though the above mechanisms are universally applicable to the genesis of all heart sounds and murmurs, for clinical purposes it is useful to consider individual sounds and the mechanism of production.

INTENSITY OF FIRST HEART SOUND

Intensity is determined by:

- Structural integrity of the mitral valve
- Position of the AV valve at the time of ventricular contraction
- Integrity of isovolumic systole
- Heart rate
- P-R interval
- Myocardial contractility

Structural integrity of mitral valve

The various components of the mitral valve make up the mitral valve apparatus (Fig. 17.1).

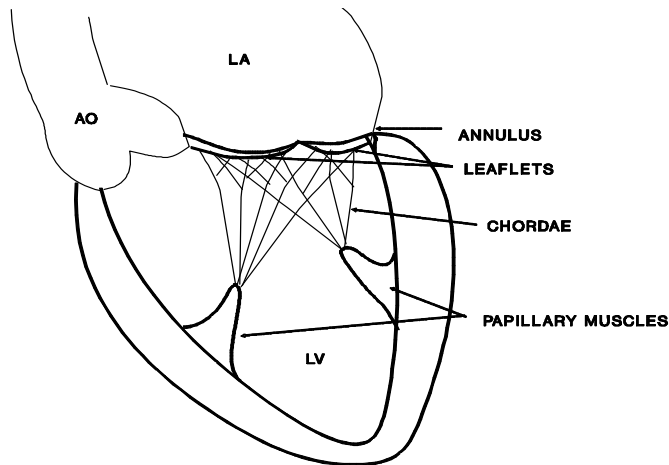


Fig. 17.1: Components of the mitral valve apparatus

The mitral valve apparatus consists of:

- Leaflets/commissures
- Chordae
- Papillary muscles
- Left ventricle
- Mitral annulus

The normal mitral valve has thin pliable leaflets, which are capable of producing a normal first heart sound. A calcified immobile valve results in diminished or absent first heart sound. Loss of leaflet tissue (as in infective endocarditis) will have a similar effect.

Position of AV valve during ventricular contraction

The normal mitral valve is in a semi-closed position by the end of the diastole as the leaflets float up in the left ventricle that is almost completely filled. The ventricular contraction closes it further, producing the normal first heart sound. When the mitral valve is open, or cannot semi-close by the end of the diastole, a loud first sound occurs as in mitral stenosis. In mitral stenosis, the mitral valve is kept open in the later phase of the diastole due to high pressure in the left atrium (Fig. 17.2). This is because, the excursion of the valve is increased and the mitral valve closes late and at a higher pressure of the left ventricle, due to left atrial hypertension. In atrial septal defect, the tricuspid component of the first heart sound is increased due to large tricuspid flow keeping the valve open. Hyperkinetic

circulatory states and left to right shunts also increase flow across the mitral valve to accentuate the first heart sound. A short P-R interval keeps the mitral valve wide open when the next ventricular contraction begins and also produces a loud first heart sound. Paradoxically, if the mitral valve is closed prematurely the first heart sound is diminished or absent.

Pressure wave forms of left ventricle and left atrium are represented to indicate the end-diastolic gradient at the onset of systole that keeps the mitral valve in open position, thus increasing the amount of excursion the valve has to undergo before it closes. This results in the loud first heart sound. Conversely a soft first heart sound is generated when the mitral valve closes prematurely. Normally, the mitral valve closes at the onset of systole as LV pressure exceeds LA pressure. However, in acute aortic regurgitation or chronic aortic regurgitation with left ventricle dysfunction, the left ventricular diastolic pressure exceeds the left atrial pressure before the onset of systole, resulting in premature closure of the mitral valve. This is very well appreciated in the M-mode echocardiogram.

The causes for **premature closure of the mitral valve** could be:

- Acute severe AR
- Chronic severe aortic regurgitation with LV dysfunction
- Prolonged PR interval

The mechanism is shown in (Fig. 17.3).

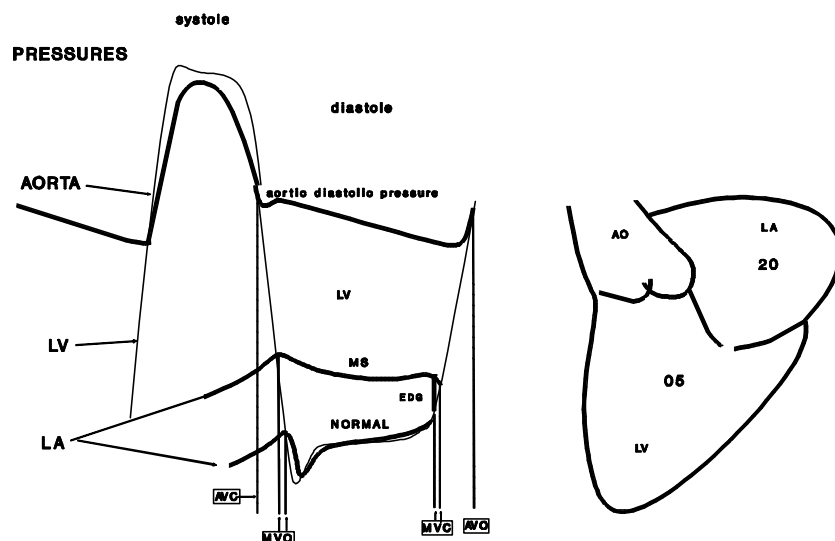


Fig. 17.2: Mechanism of loud first heart sound in mitral stenosis

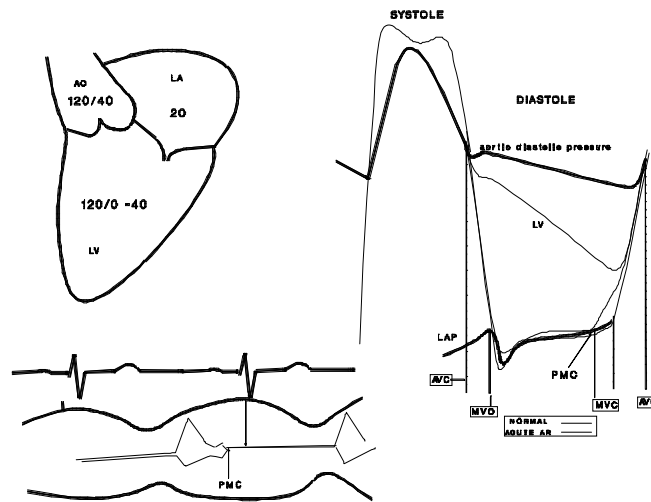


Fig. 17.3: Mechanism of premature closure of mitral valve in aortic regurgitation

Integrity of isovolumic systole

Normally, during the phase of isovolumic systole, the ventricle contracts like a closed chamber, due to integrity of the ventricular muscle, mitral valve and the aortic valve. During this phase the pressure rises steeply and the rate of rise of pressure (dp/dt) is maximum. When isovolumic systole is lost or abbreviated due to any cause (Fig. 17.4), the rate of rise of left ventricle pressure (dp/dt) falls steeply resulting in decreased velocity of closure of the mitral valve. The first heart sound is diminished or absent when isometric contraction is compromised as in severe mitral regurgitation, aortic regurgitation or in ventricular aneurysm. In mitral regurgitation, ventricular aneurysm with dyskinesia, the isovolumic systole is lost (as shown in Fig. 17.4), whereas, in aortic regurgitation, it is abbreviated due to very low diastolic pressure of aorta. However, in ventricular septal defect, in spite of loss of isovolumic systole the first sound is not diminished as often and is possibly related to the two ventricles behaving like a common chamber.

The causes of loss of isovolumic systole are:

- Severe mitral regurgitation
- Severe aortic regurgitation
- Large ventricular septal defect
- Ventricular aneurysm

THE FIRST HEART SOUND

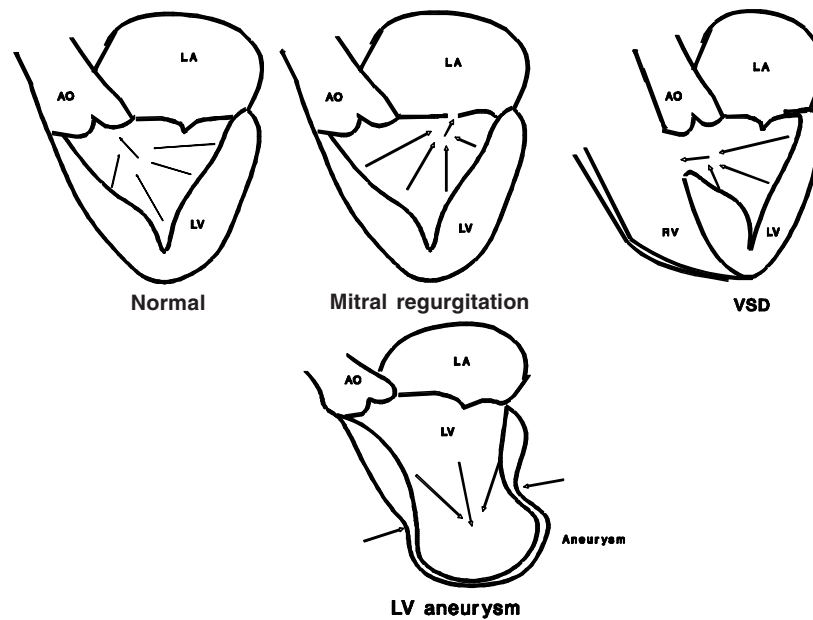


Fig. 17.4: Loss of isovolumic systole

Normally, the mitral valve closes at the onset of systole as left ventricle pressure exceeds LA pressure (Fig. 17.4). However, in acute aortic regurgitation or chronic aortic regurgitation with left ventricle dysfunction, the left ventricular diastolic pressure exceeds the left atrial pressure before the onset of systole, resulting in premature closure of the mitral valve. This is very well appreciated in the M-mode echocardiogram.

Heart rate

Tachycardia accentuates first heart sound by shortening the P-R interval, increase in contractility, and a wide open valve due to an abbreviated diastole. Bradycardia has the opposite influence.

P-R interval

The short P-R interval increases first heart sound by the mechanism of wide open valve as the atrial contraction occurs in tandem with ventricular contraction. A prolonged P-R interval results in diminished first heart sound due to premature closure of the mitral leaflet.

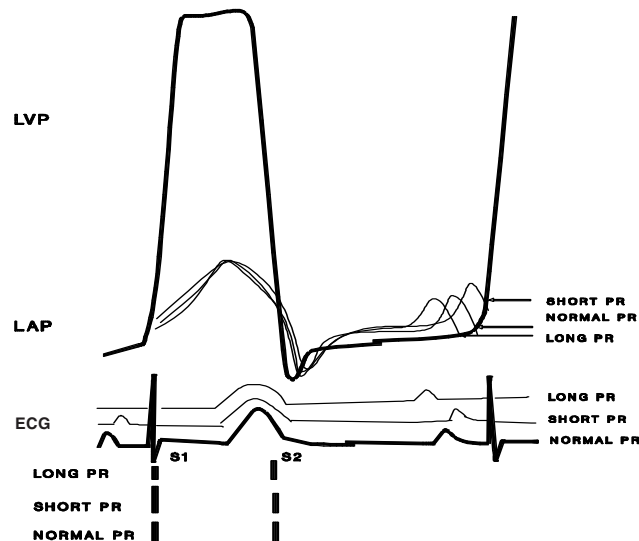


Fig. 17.5: Relationship of P-R interval to the intensity of first heart sound – intracardiac pressure mechanics

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The P-R interval influences the intensity of the first heart sound due to the relationship of atrial contraction to the onset of systole (Fig. 17.5). When the P-R is short, closely occurring atrial contraction keeps the mitral valve open at the onset of systole, thereby increasing first heart sound intensity.

Myocardial contractility

Increase in myocardial contractility increases the first heart sound; diminished contractility has a negative influence.

The causes of increased myocardial contractility could be:

- Exercise
- Emotional excitability
- Hypoglycemia
- Thyrotoxicosis
- Pheochromocytoma
- Drugs: sympathomimetics, β_2 stimulants

The causes of diminished myocardial contractility could be:

- Myocardial ischemia or infarction
- Myocarditis

THE FIRST HEART SOUND

- Cardiomyopathy
- Ventricular dysfunction due to any cause
- Drug induced myocardial depression
- Betablockers, verapamil, diltiazem, disopyramide

The normal first heart sound is a relatively prolonged, low frequency sound having two components, and is best heard at the apex. The split of the first heart sound is heard only at the tricuspid area, as the tricuspid component is heard only at this site.

EVALUATION OF FIRST HEART SOUND

The first heart sound has to be evaluated along the following parameters:

- Intensity
 - Normal
 - Accentuated
 - Diminished
 - Constancy or variability
- Split

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For example, when the first sound is normal it is expressed as follows: 'The first sound is normal in intensity, split is normal and is constant in intensity'. The split of the first heart sound does not have the same significance as that of second heart sound and is heard in only 40 per cent of normal individuals.

Intensity

Normally, the first heart sound should be loudest at the apex and the second heart sound should be louder at the base. If the first heart sound is equal to, or higher in intensity than the second heart sound at the base, the first heart sound is considered accentuated (Table 17.1). Diminution of the first heart sound is based on subjective evaluation (Table 17.2). Further the concept of 'normal' is itself relative. As this evaluation is subjective, mild alterations in intensity should not be used to rule in or rule out a disorder. With a normal heart rate and P-R interval, if the first heart sound is loud one should consider mitral stenosis.

Variable intensity

The normal first heart sound is constant in intensity because most of the determinants of the intensity of first heart sound are constant over a period of

Table 17.1: Causes and mechanisms of loud first heart sound

<i>Causes</i>	<i>Mechanisms</i>
Exercise	Tachycardia Shortened P-R interval Increased myocardial contractility Increased flow across AV valves
Emotional excitability	As above Catecholamines
Mitral stenosis	Wide open mitral valve at end diastole Delayed mitral valve closure Mitral closure at higher left ventricle pressure Thickened but mobile mitral leaflets
Hyperkinetic circulatory states	Increased AV valve flow Increased myocardial contractility Tachycardia
Atrial septal defect	Increased tricuspid flow
Sinus tachycardia	Shortened diastole Wide open mitral valve Increased myocardial contractility (Treppe phenomenon) Short P-R interval
Short P-R interval	Wide open AV valves

time. Certain features like valve anatomy cannot vary, but others like heart rate, P-R interval, position of the AV valve by the end of diastole, and force of ventricular contraction can vary. In eliciting constancy or variability in the intensity of first heart sound, the patient should be asked to hold breath to avoid respiratory alteration in the intensity of first heart sound.

The causes could be:

- Atrial fibrillation
- Complete heart block
- Ventricular tachycardia
- Classic atrioventricular dissociation (isorhythmic AV dissociation)
- Atrial flutter with varying block
- Atrial tachycardia with varying block
- Multifocal atrial tachycardia
- Frequent atrial and ventricular ectopy

THE FIRST HEART SOUND

Table 17.2: Causes and mechanisms of diminished first sound

<i>Cause</i>	<i>Mechanisms</i>
Sinus bradycardia	Long diastolic filling allows premature closure of AV valve Due to reverse of Treppe phenomenon there can be diminished contractility in sinus bradycardia leading to diminished first sound
Prolonged P-R interval	Premature mitral valve closure
Severe mitral regurgitation	Loss of isovolumic systole Failure of leaflets to close Fibrosis shortening and diminished mobility of valve
Chronic severe AR	Loss of isovolumic systole Premature closure of MV (rare)
Acute severe AR	Premature closure of MV (common) Loss of isovolumic systole
Ventricular aneurysm	Loss of isovolumic systole Diminished ventricular contractility
Acute myocardial infarction	Diminished ventricular contractility Prolonged PR interval LBBB Mitral regurgitation Ventricular aneurysm
Myocarditis	Loss of contractility
Cardiomyopathy	As above and under myocardial infarction
Calcific MS	Immobile mitral valve
Left bundle branch block	Asynchronous ventricular activation
Betablocker therapy	Depressed ventricular function Bradycardia

In atrial fibrillation variation in diastolic cycle lengths results in variable position of the AV valve by the end of the diastole (Fig. 17.6); the force of ventricular contraction is also variable due to the differing diastolic volumes of the ventricle. In complete heart block, ventricular tachycardia and classic AV dissociation, variability in first sound due to the variability of the P-R interval. In mitral stenosis, in spite of atrial fibrillation, the first heart sound may not vary significantly due to a very loud first heart sound. The valve is generally kept open irrespective of the diastolic cycle lengths due to high left atrial pressures.

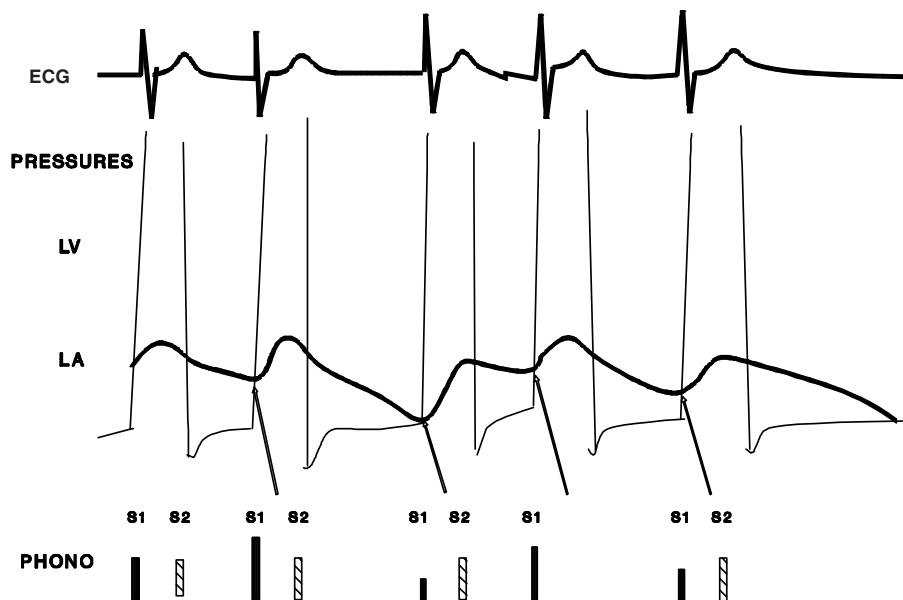


Fig. 17.6: Mechanism of varying intensity of first heart sound in atrial fibrillation

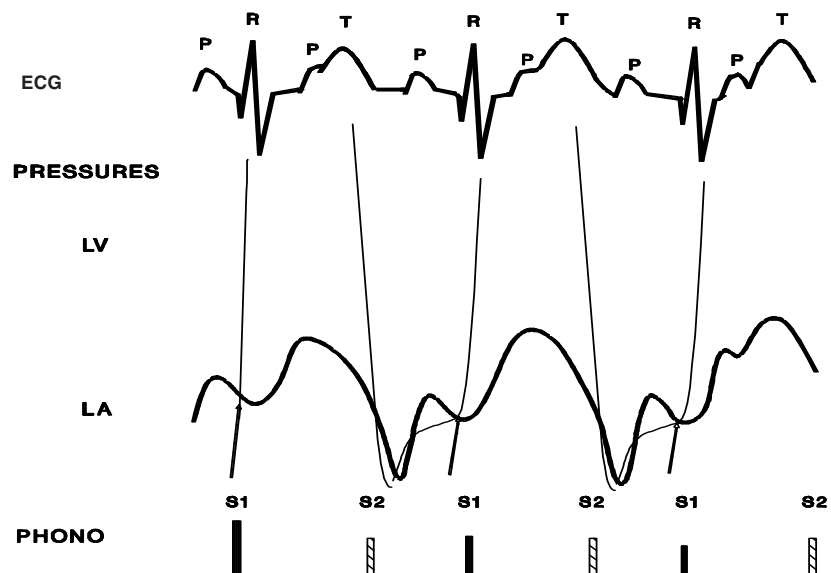


Fig. 17.7: Mechanism of varying first heart sound in complete heart block

THE FIRST HEART SOUND

In complete heart block, due to dissociation of atrial and ventricular activation, the atria and ventricles contract independently. When the atria contract just before the ventricular contraction, the first heart sound tends to be loud (Fig. 17.7). Similarly, if the atria contract much before ventricular activation, the first heart sound tends to be softer.

Similar to complete heart block, there may be atrioventricular dissociation in junctional tachycardia resulting in varying intensity of first heart sound (Fig. 17.8).

Varying diastolic intervals result in differing force of ventricular contraction, which also determines the intensity of first heart sound (Fig. 17.9). The atrioventricular dissociation also contributes to the varying first heart sound.

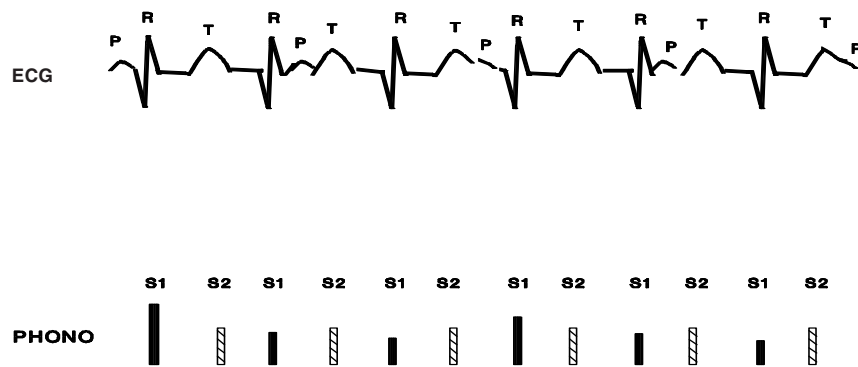


Fig. 17.8: Mechanism of varying intensity of first heart sound in AV dissociation

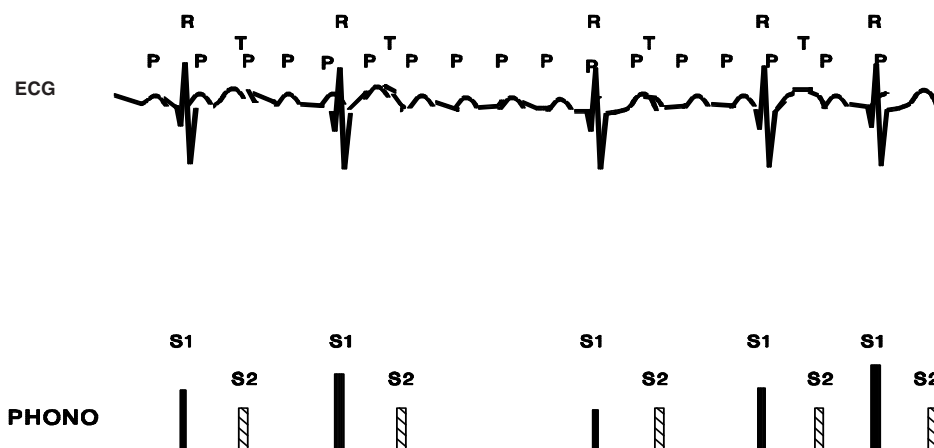


Fig. 17.9: Mechanism of varying intensity in atrial flutter with variable block

Mechanism and significance of varying first sound in ventricular tachycardia

Variation in S1 intensity, irregular cannon waves in jugular venous pulse, and variation in arterial pressure are well known signs of ventricular tachycardia. In a recent study, Garratt et al analyzed systematically the value of physical signs in the bedside diagnosis of ventricular tachycardia. It has been shown that variable intensity of S1 and jugular venous pulse are highly specific and sensitive in the diagnosis of ventriculoatrial dissociation during ventricular tachycardia. The variability of arterial pulse volume was not very helpful.

First heart sound split

The causes of a wide split first sound could be:

- Right bundle branch block
- Atrial septal defect
- Ebstein's anomaly of tricuspid valve

FIRST HEART SOUND IN VARIOUS CLINICAL STATES**MITRAL STENOSIS**

The loud first heart sound in mitral stenosis calls attention to the possible presence of mitral stenosis, as this sign is the easiest to detect. The mid-diastolic murmur and opening snap are not as easily detectable. With calcification or severe subvalvular fusion the sound is reduced or altogether absent. The intensity of first heart sound is no guide to the severity of mitral stenosis but may help in assessing the pliability.

Mechanism of loud first heart sound

The mechanisms of a loud first heart sound in mitral stenosis are:

- Open mitral valve at end diastole
- Delayed closure of mitral valve
- Mitral valve closure at higher pressure of left ventricle
- Thickened but mobile mitral valve

The thickened leaflets and high pressure in the left atrium are the central features.

Causes of soft first heart sound

The causes of normal or diminished first heart sound in mitral stenosis are:

- Heavily calcified mitral valve
- Severe subvalvular fusion
- Associated mitral regurgitation
- Associated severe aortic regurgitation
- Masked left ventricular events due to severe right ventricular hypertrophy

The aortic ejection click is often mistaken for a loud first heart sound. When the first heart sound is louder at the base than the apex, an ejection click is likely.

MITRAL REGURGITATION

The first sound is diminished or absent in mitral regurgitation depending on the severity of mitral regurgitation. In mild mitral regurgitation, the first heart sound is normal but is diminished or absent in moderate to severe mitral regurgitation. The loss of isovolumic systole and lack of leaflet apposition may be responsible for reduction in the intensity of the first heart sound.

Mechanism

The mechanisms of reduced first heart sound in mitral regurgitation are:

- Loss of isovolumic systole
- Fibrosis and shortening of leaflets
- Failure of leaflets to close
- Myocardial dysfunction as in secondary mitral regurgitation

Causes of loud first heart sound

If the first heart sound is loud, mitral regurgitation is unlikely and one of the following conditions is likely:

- Loud ejection click of AS mistaken for first heart sound
- Severe TR of silent mitral stenosis, mistaken for mitral regurgitation
- Associated mitral stenosis
- Mitral regurgitation of MVP
- Rheumatic mitral regurgitation with well preserved anterior leaflet

When aortic stenosis is mistaken for mitral regurgitation, the accompanying ejection click will be mistaken for a loud first heart sound. In mitral valve prolapse

when the prolapse occurs early the click simulates a loud first sound. In some children with rheumatic mitral regurgitation, the first heart sound is loud with a well preserved anterior leaflet. This has implications in management as these valves are amenable to repair of the valve and mitral valve replacement can be avoided. The first heart sound is diminished out of proportion to the severity of mitral regurgitation in papillary muscle dysfunction of coronary artery disease and functional mitral regurgitation of cardiomyopathy.

The causes of milder mitral regurgitation with diminished first heart sound are:

- Mitral regurgitation in coronary artery disease
- Papillary muscle dysfunction
- Severe ventricular dysfunction
- Accompanying first degree AV block
- Left bundle branch block
- Secondary mitral regurgitation in cardiomyopathies
- Associated severe aortic regurgitation

The basic underlying mechanism in all the above conditions is severe left ventricular dysfunction which by itself decreases the intensity of S1.

PRACTICE IMPLICATIONS

- ➔ Loud S1 with normal heart rate and P-R interval could be indicative of mitral stenosis.
- ➔ The intensity of S1 has no correlation to the severity of mitral stenosis.
- ➔ A loud S1 does not rule out calcified mitral valve.
- ➔ Variable intensity of S1 is a very reliable sign of ventricular tachycardia and is helpful in distinguishing it from supraventricular tachycardia with aberration. This sign is more useful than some of the electrocardiographic signs.

18 The Second Heart Sound

The second heart sound is due to closure and vibrations of the semilunar valves. The aortic component is louder and earlier than the pulmonary component. The aortic sound is louder due to the higher pressure in the aorta. The aortic sound occurs earlier and the pulmonic sound later, because of the pressures the valves face and the ejection properties of the two ventricles. The pressures in the aorta are higher and the left ventricular ejection is finished earlier. Additionally, the hangout interval plays an important role.

Hangout interval

By the end of ventricular ejection, the ventricular pressures start falling and are lower than the arterial pressures.

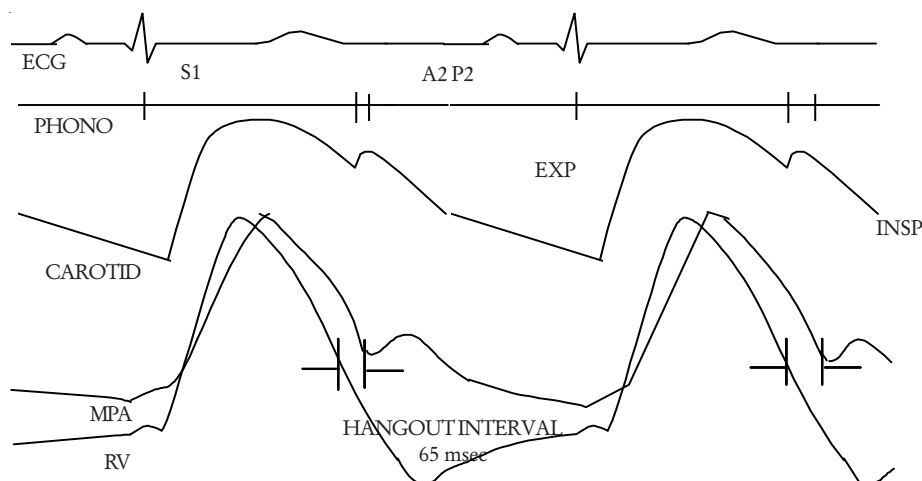


Fig. 18.1: Pressure wave form depiction of hangout interval on the right side

The semilunar valve is expected to close at the point of crossover of pressures (the point where the ventricular pressure falls lower than the arterial pressure). However, in reality, the semilunar valve closure does not occur at the time of crossover of pressures but slightly later. This time interval from the crossover of pressures to the actual occurrence of sound is called the hangout interval (Fig. 18.1). The hangout interval is related to the pressures in the artery, distensibility and the elastic recoil of the arterial system. Due to higher pressure and less distensibility, the hangout interval on the aortic side is approximately 30 msec compared to 80 msec on the pulmonary side. The factors influencing hangout intervals are:

- Pressure beyond the valve
- Dilatation of the artery
- Distensibility of the arterial system
- Vascular impedance
- Phase of respiration

In reality, all these factors are interrelated. Inspiration, by stretching the pulmonary vasculature, increases the hangout interval of pulmonary circulation.

EVALUATION OF SECOND HEART SOUND

The features used for evaluating the second heart sound are the split and the intensity of the two components.

SPLIT

Normal split

The normal second heart sound is split into two components during inspiration and is single during expiration (Fig. 18.2). During inspiration, we not only inspire air into the lungs, but also draw in blood into the thorax, secondary to a fall in intrathoracic pressure.

This inspiratory increase in venous return increases the stroke volume of the right ventricle prolonging RV ejection. This postpones the pulmonic sound to a later time in the cardiac cycle. The inspiratory decrease in venous return to the left ventricle reduces the left ventricular stroke volume and shortens the left ventricular ejection. This advances the aortic sound to an earlier time in cardiac cycle.

THE SECOND HEART SOUND

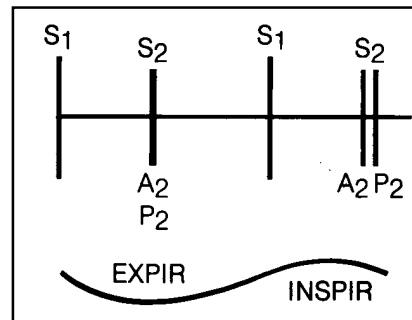


Fig. 18.2: Normal splitting of S2 with respiration

Abnormal split

Two features describe the characteristics of second heart sound splitting: the width, and the movement with respiration. The determinants of a normal split are:

- Pressure difference between the two circulations
- Different ejection properties of the two ventricles
- Difference in the hangout interval in the aorta and pulmonary artery
- The right and left sided venous returns separated by the interatrial septum
- Ability of respiration to alter the above factors
- The simultaneous onset of electrical impulse to either ventricle

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The possible abnormalities are:

- No split or single second heart sound
- Wide split variable
- Wide split fixed
- Reversed or paradoxical split

The single second heart sound: The single second sound by definition means absence of an audible split in either phase of respiration.

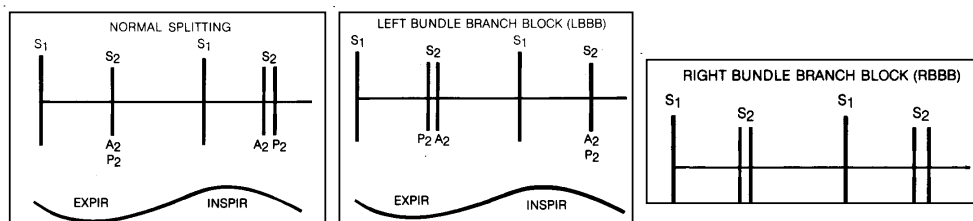


Fig. 18.3: Definitions of splitting of second heart sound (S2): normal split, reversed split in LBBB, wide split in RBBB

Table 18.1: Mechanisms of single second sound

<i>Mechanism</i>	<i>Disease</i>
Only one semilunar valve is present	Truncus arteriosus
One of the semilunar valves is atretic	Pulmonary atresia Aortic atresia
Posterior location of pulmonary valve	Transposition of great vessels
Severe stenosis of one semilunar valve	AS or PS, with or without calcification
Extreme loudness of one of the sounds	Single loud P2 in extreme PAH

In severe pulmonary arterial hypertension, the pulmonic sound is extremely loud and masks the preceding aortic sound by the phenomenon of retrograde masking.

To recognize a single second heart sound one must auscultate carefully in either phase of respiration, supine position with passive leg raising, during the post-release phase of Valsalva, and tilting the patient upside down (in case of infants). Normally the second heart sound split is audible only in the pulmonary area unless the pulmonic sound is accentuated.

The detection of a single second heart sound is of diagnostic and prognostic significance in various clinical settings.

The single second sound is due to

- Normal variant as in elderly people
- Congenital heart disease
- Acyanotic
- Severe AS especially calcific
- Severe PS
- Ventricular septal defect with PAH

Wide split second sound: The wide split by definition means an audible split in expiration. This definition is qualitative and not quantitative. Theoretically, a wide split can occur whenever the pulmonic sound occurs later or the aortic sound occurs earlier (Fig. 18.4).

The normal A2-P2 interval ranges between 20 and 50 msec in inspiration. It varies by > 20 msec with expiration. When the split is more than 50 msec it is easily appreciated as a wide split. Normally, the split is not detectable in standing position, in expiration as the split is < 15 msec, which cannot be heard by the

THE SECOND HEART SOUND

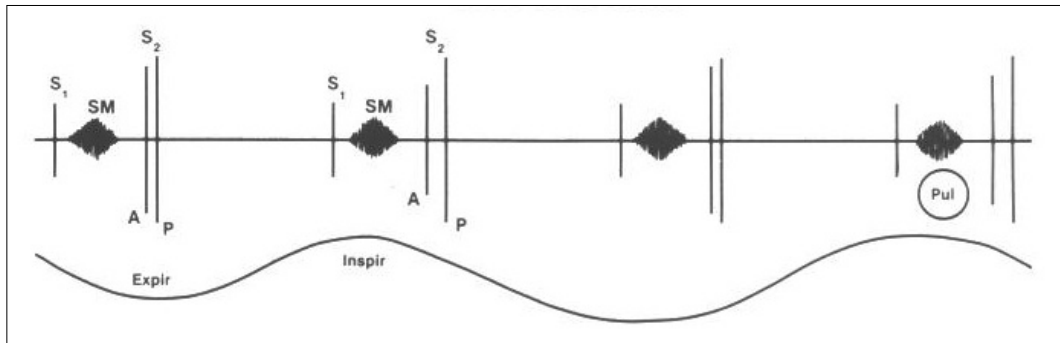


Fig. 18.4: Wide splitting of second heart sound

human ear. Clinically, the split is defined as wide, if it is heard well in standing position, in expiration.

The pulmonic sound can occur later when there is a delay in the onset of electrical impulse to the RV or prolongation of right ventricular ejection (Table 18.2). The aortic sound may occur earlier than normal when the left ventricular ejection is finished earlier (than normal).

Table 18.2: Mechanisms and causes of wide split second heart sound

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<i>Mechanisms</i>	<i>Causes</i>
Prolonged RV ejection	Moderate to severe PS Severe pulmonary arterial hypertension Acute pulmonary embolism ASD (large RV stroke volume) Severe right ventricular failure
Delayed electrical impulse to RV	RBBB LV pacing (epicardial pacing) LV ectopy
Increase in hangout interval	Normal variant Idiopathic dilatation of pulmonary artery ASD Postoperative ASD Pulmonic stenosis with post-stenotic dilatation
Earlier completion of LV ejection	Severe mitral regurgitation
Extreme delay in aortic sound	Reversed wide split
Impaired diastolic filling	Restrictive cardiomyopathy Hypertrophic disorders of myocardium Constrictive pericarditis

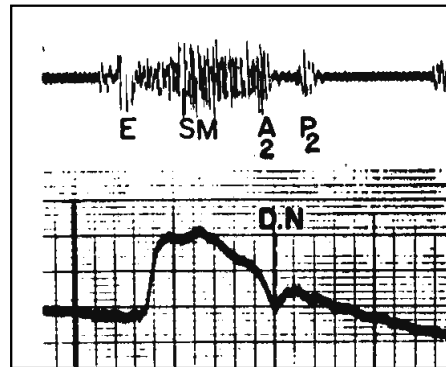


Fig. 18.5: Wide split

S2: in severe valvular pulmonary stenosis E: ejection click, SM: systolic murmur, A2: aortic component of second sound, P2: pulmonic component of second sound, DN: dicotic notch.

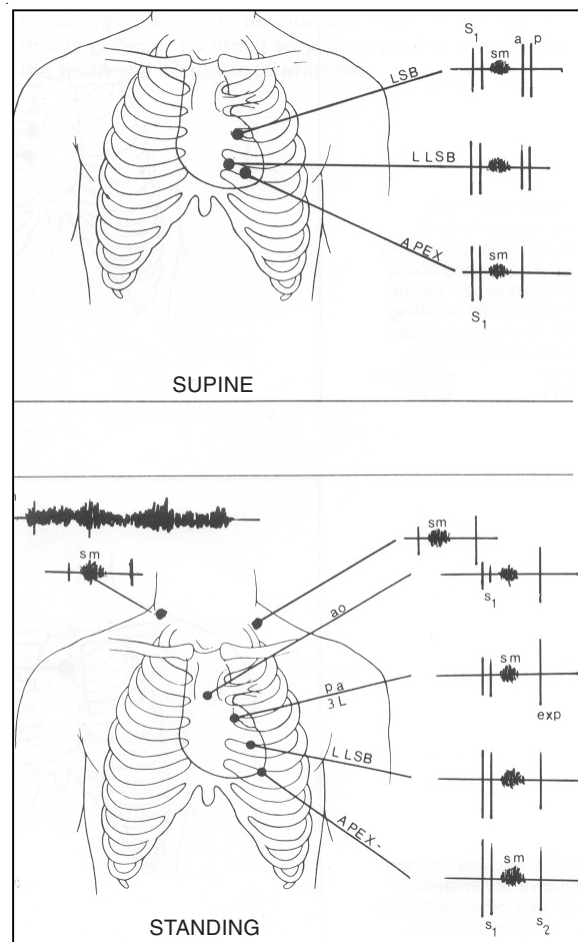


Fig. 18.6: Normal split that is audible in supine position disappears in standing position

THE SECOND HEART SOUND

In conditions with diastolic dysfunction, a change in filling pressure evokes a smaller change in cardiac dimension. This results in lesser alteration in systolic performance with respiration causing 'fixed' or near fixed split of second heart sound.

Some of the conditions mentioned above are associated with a 'wide and fixed split'. The split is considered as 'fixed' if the two components fail to move with respiration. The normal second heart sound split varies because of the ability of respirations to vary the venous return to either side of the heart

The variability of the second heart sound split could be due to:

- The two atria separated by interatrial septum
- Ability of respirations to alter venous return to either side of heart
- A competent non-failing right ventricle capable of increasing the output by the increase in stroke volume

Once the reasons for the variability of the split is known, the mechanisms of a 'fixed' split are easy to understand.

<i>Causes of wide and 'fixed' split</i>	<i>Mechanisms of 'fixed' split</i>
Atrial septal defect	Defect in the interatrial septum allowing free communication between the two atria
All the causes of wide split with associated severe right ventricular failure	Right ventricle failing to increase the stroke volume from the increased venous return

Recognizing a wide and 'fixed' split: An audible expiratory split during standing always means a wide split. Some normal children have an expiratory split in supine position. In case of any doubt, the persistence of the split during the straining phase of Valsalva can be looked for.

Reversed split: The reversed split by definition means an inaudible split during inspiration and an audible split during expiration. A reversed split may occur due to delayed occurrence of aortic sound or an early pulmonic sound. The mechanisms of delayed occurrence of aortic sound may be electrical or mechanical. Earlier than normal occurrence of pulmonic sound is generally due to an electrical mechanism (Table 18.3).

Normally, the aortic sound precedes the pulmonic sound in both the phases of respiration. When the pulmonic sound precedes aortic sound it is called a *reversed split*. If the reversal is confined to expiration, it is called *partial reversed split*. Clinically,

Table 18.3: Mechanisms and causes of reversed splitting of second heart sound

<i>Mechanisms</i>	<i>Causes</i>
Delayed electrical activation of LV	LBBB RV pacing RV ectopy
Prolonged LV mechanical systole	Severe AS Severe systemic hypertension Acute myocardial infarction During an episode of angina Cardiomyopathy Severe AR Large patent ductus arteriosus
Increase of hangout interval on the aortic side	Aneurysm of ascending aorta Post-stenotic dilatation in AS
Early pulmonic closure	Early electrical activation of RV as in type B WPW syndrome. In WPW syndrome, reversed split occurs only when there is significant pre-excitation. Severe tricuspid regurgitation

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the reversed split is recognized by a wider split in expiration and often can be mistaken for a wide normal split.

Clinical recognition of reversed split: Based on the degree of delay in A₂, the spectrum of reversed split varies from a single second heart sound to a frankly audible paradoxical split (Fig 18.7). When the split is audible in expiration and not in inspiration, it is fairly easy to recognize the reversed split.

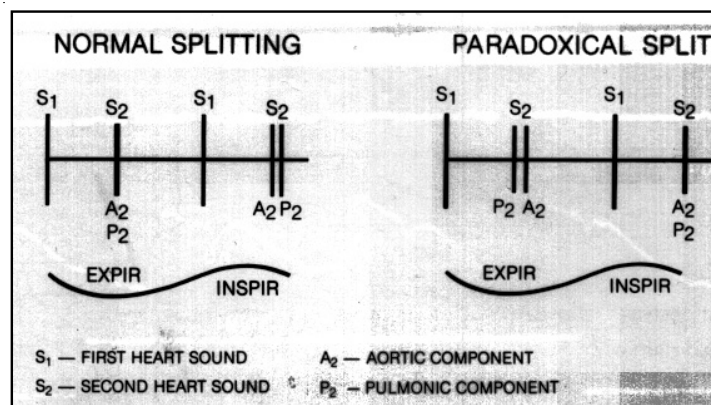


Fig. 18.7: Paradoxical splitting of second sound, left bundle branch block depicted on right side

THE SECOND HEART SOUND

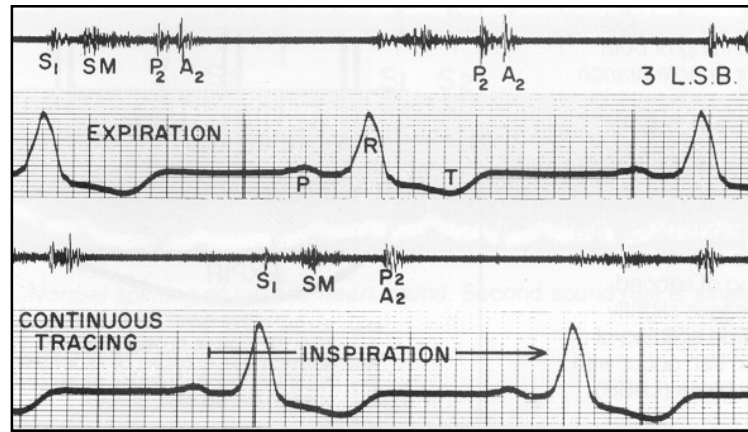


Fig. 18.8: Reversed splitting of second heart sound

The spectrum of reversed split is:

- A single second heart sound
- Incomplete reversed split
- Complete reversed split
- Reversed wide split variable
- Reversed wide split 'fixed'

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In actual practice the reversed split is often confused for a wide and variable split or a wide and fixed split. This is because in inspiration the split may narrow or may remain the same when there is a left ventricular dysfunction in association with aortic stenosis or left bundle branch block. The best way to appreciate the reversed split is to trace the two components of the second heart sound away from the pulmonic area to the apex. Normally only the earlier of the two components (aortic sound) is traced to the apex and the last component is not heard away from the pulmonary area. When the 'later' of the two components is traceable to the apex a reversed split is likely.

INTENSITY

After evaluation of the split, the intensity of the two components of second heart sound should be stated. As the mechanism of the closure of the atrioventricular valves are different from that of the semilunar valves, the aortic sound and pulmonic sound are influenced differently from S1. The intensity is influenced by:

- Pressure beyond the valve

- Flow across the valve
- Size of the vessel beyond the valve
- Stenosis of the valve
- Regurgitation of the valve

When the pressure beyond the valve is elevated, as in systemic or pulmonary hypertension, the corresponding second sound is increased. When the flow across the pulmonic valve is increased as in left to right shunts, the pulmonic component is accentuated. The flow across the aortic valve is increased in hyperkinetic states and aortic regurgitation. Dilatation of the vessel beyond the valve as seen in pulmonary hypertension, left to right shunts and idiopathic dilatation of the pulmonary artery also increases the pulmonic sound. The aortic component is similarly affected when the ascending aorta is dilated as in aneurysm of the ascending aorta. Stenosis of the semilunar valve with attendant restriction of valve mobility, reduces the intensity of the corresponding second heart sound.

Whether the aortic sound is accentuated or diminished in aortic regurgitation, depends on the mechanism of the regurgitation (Table 18.4). In aortic regurgitation due to aortic root disease, as in syphilis or ankylosing spondylitis, the aortic sound is usually accentuated. In aortic regurgitation due to aortic valve disease, as in rheumatic heart disease, the aortic sound is diminished or absent due to a fibrous and immobile valve. The aortic sound is often sharper than normal in congenital bicuspid aortic valve as long as the valve is not significantly stenosed.

When the pulmonic sound is extremely loud and banging as in severe pulmonary arterial hypertension, the preceding aortic sound cannot be heard due to retrograde masking (Tables 18.5 and 18.6).

Table 18.4: Causes and mechanism of accentuated aortic sound

<i>Causes</i>	<i>Mechanisms</i>
Systemic hypertension	Elevated pressure beyond the valve Dilated ascending aorta
Aneurysm of ascending aorta	Dilatation of vessel
Aortic regurgitation	Aortic root disease Well preserved leaflet mobility Increased flow across the valve Dilated ascending aorta
Congenital bicuspid aortic valve	Thickened but mobile aortic leaflets

THE SECOND HEART SOUND

Table 18.5: Loud pulmonic sound

<i>Causes</i>	<i>Mechanisms</i>
Normal in infants and children	Higher pulmonary arterial pressure
Adults with chest deformity or thin chests	Proximity of PA to the stethoscope
Pulmonary arterial hypertension	Higher closing pressure of valve Dilated PA
Left to right shunts	Increased flow across the valve with exaggerated valve excursion Dilated PA PAH
Hyperkinetic circulatory states	Increased flow across the valve with exaggerated valve excursion Dilated PA

Table 18.6: Diminished pulmonic sound

<i>Causes</i>	<i>Mechanisms</i>
<i>Diminished</i> Normal in elderly Thick chested adults Pulmonary stenosis Dysplastic valve	Diminished valve excursion
<i>Absent</i> Tetralogy of Fallot Transposition of great arteries Truncus arteriosus Pulmonary atresia Absent pulmonary valve	Severe pulmonic stenosis Pulmonary artery posteriorly located Pulmonary valve is absent

Clinical evaluation

Normally, in the pulmonary area both components of the second heart sound are audible and the aortic component is louder. The pulmonic component is localized to the pulmonary area and the aortic component is widely audible in other areas. Even in the pulmonary area, the aortic component is louder than the pulmonary component. The pulmonic sound is considered accentuated if it is equal to the aortic sound in the pulmonary area. Accentuation of pulmonic sound is graded as mild (if it is equal to the aortic sound), moderate (if it exceeds aortic sound) and severe (if it is extremely loud and banging). It is also expressed, respectively, as (+), (++) and (+++).

Table 18.7: Grading and correlations of pulmonic sound intensity

<i>Grading of pulmonic sound</i>	<i>Basis</i>	<i>Correlation to PA pressure</i>
Normal	Pulmonic sound less than A2	Normal PA pressures Systolic < 30 Mean 20
Mild or +	Pulmonic sound equal to A2	Mild PAH Systolic 30–35 Mean 20–30
Moderate or ++	Louder than A2	Moderate PAH Systolic 40–75; Mean 30–50
Severe or +++	Very loud/banging	Severe PAH Systolic > 75 Mean > 50

In mild pulmonary hypertension (PAH), the pulmonic sound is equal in intensity to the aortic sound; in moderate pulmonary hypertension, the pulmonic sound is louder than the aortic sound; and in severe pulmonary hypertension, the pulmonic sound is very loud and banging. Other variables like chest wall thickness or emphysema, might influence the correlations.

When the aortic sound is diminished as in aortic stenosis the audibility of the pulmonic sound away from the pulmonary area, is helpful in its evaluation. The grading of intensity of pulmonic sound as mild, moderate and severe is helpful as it correlates well with similar degrees of pulmonary arterial hypertension. Exceptions to this are patients with thin chest wall, chest deformities and children (where the pulmonic sound can be loud as a normal variant). The intensity of the pulmonic sound is underestimated in patients with thick chest wall, emphysema with chronic cor pulmonale and some patients with primary pulmonary hypertension and severe heart failure.

NORMAL VERSUS ABNORMAL HEART

In children and adolescents, systolic murmurs along the left sternal border and pulmonary area are common. The differential diagnosis often involves an innocent systolic murmur, straight back syndrome, atrial septal defect, or pulmonic stenosis. If the second sound split is normal, the first two possibilities are likely.

THE SECOND HEART SOUND

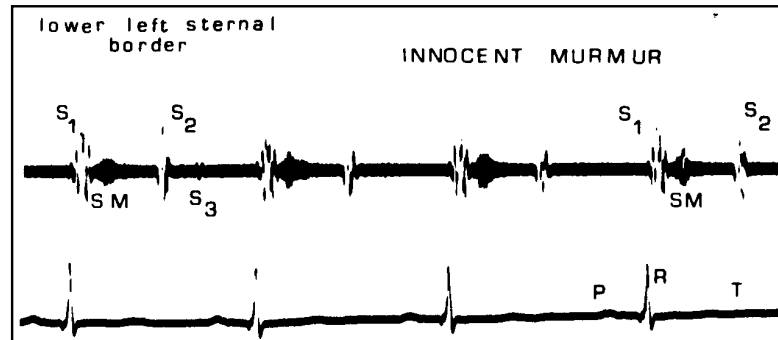


Fig. 18.9: Innocent systolic murmur, physiological S3, normal split of S2

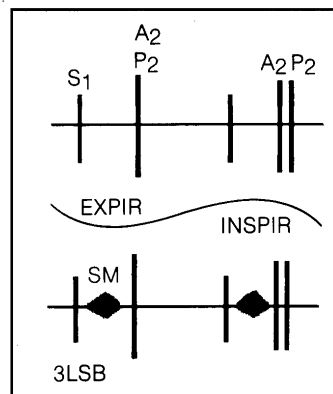


Fig. 18.10: Innocent systolic murmur in a child

The innocent systolic murmur in children is often accompanied by a physiological third sound as shown in Figs. 18.9 and 18.10.

CONGENITAL HEART DISEASE

ATRIAL SEPTAL DEFECT

The second heart sound is wide and fixed. The pulmonic sound may be loud in atrial septal defect (ASD) even in the absence of pulmonary arterial hypertension due to increased flow across the pulmonic valve with a dilated pulmonary artery (Fig. 18.11).

If the split varies with respiration in an atrial septal defect, either an ASD is unlikely or is too small, or partial anomalous pulmonary venous drainage is present. Even after the advent of Eisenmenger syndrome, the wide split continues to be heard in atrial septal defect (Fig. 18.12).

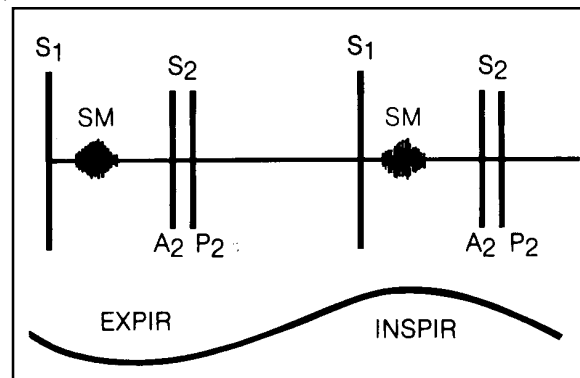


Fig. 18.11: Phonocardiogram in a patient with atrial septal defect

	INSPIRATION			EXPIRATION		
	S1	A	P	S1	A	P
ASD - NO PAH						
ASD WITH PAH						
ASD - EISENMENGER'S						
ASD WITH PS						
SMALL ASD or PAPVC						

Fig. 18.12: Second heart sound in atrial septal defect

Typically, the split is wide and fixed with accentuated pulmonic sound, which becomes intensifies with pulmonary hypertension. When Eisenmenger syndrome develops, the split is still wide and fixed with a banging pulmonic sound. ASD associated with pulmonic stenosis results in further widening of the split with diminution in pulmonic sound intensity. The split can be mobile if there is a restrictive atrial septal defect.

VENTRICULAR SEPTAL DEFECT

In small ventricular septal defect (VSD), the split is normal with a normal intensity of pulmonic sound. In moderate ventricular septal defect with left to right shunt,

THE SECOND HEART SOUND

the split is normal with moderate accentuation of pulmonic sound. In a large ventricular septal defect with left to right shunt the split is close or single. Once pulmonary arterial hypertension develops with a balanced or right to left shunt (Eisenmenger syndrome) the second sound is single as a rule. If the second heart sound is wide split in a patient with ventricular septal defect, an AV canal ventricular septal defect or associated atrial septal defect should be considered. Once the stage of Eisenmenger syndrome is reached, the second sound is single. A wide split second sound in an Eisenmenger ventricular septal defect indicates an underlying AV canal ventricular septal defect. Pulmonic sound is diminished with associated pulmonic stenosis (TOF physiology).

The aortic component of the second heart sound is usually normal in ventricular septal defect. An unusually loud A2 in VSD should indicate the possibility of the existence of a condition other than a simple VSD.

Table 18.8: Second heart sound in various subsets of ventricular septal defect

<i>VSD subset</i>	<i>Second heart sound split</i>	<i>Mechanism</i>
Small VSD	Normal P2 normal	Normal PA pressures Normal hangout interval
Moderate VSD	Normal or wide split P2 moderate intensity	Moderate PAH
Large VSD	Close split or single S2 P2 severe in intensity	PA pressures near systemic range
AV canal VSD	Wide split	Associated RBBB, ASD or MR Identical pressures
Eisenmenger VSD	Single S2 as loud P2	Equalization of hangout interval in both circulations
VSD as a part of a complex defect like TOF, TGA, or DORV	Single loud A2	Pulmonary stenosis Posteriorly located PA
VSD with coarctation of aorta, unruptured or ruptured sinus of Valsalva, bicuspid aortic valve	Loud A2	Systemic hypertension Dilated aortic sinus Thickened but mobile valve

Diagnostic implications of loud A2 in VSD

- Associated coarctation of aorta
- PDA mistaken for VSD when the diastolic component of the continuous murmur is inaudible or absent
- Bicuspid aortic valve
- Transposition of great vessels with VSD
- Systemic hypertension
- Unruptured or ruptured sinus of Valsalva with VSD
- Associated aortic regurgitation

Coexistence of any of the above conditions with VSD modifies the management of a patient with VSD (Table 18.8).

PATENT DUCTUS ARTERIOSUS

The second heart sound is normally split in patent ductus arteriosus of different sizes and remains so even after the development of severe PAH with Eisenmenger syndrome. The split is usually difficult to hear due to the continuous murmur covering the second heart sound (Fig. 18.13).

PULMONIC STENOSIS

The second heart sound is normally split in mild pulmonic stenosis. Once post-stenotic dilatation occurs, the split can be wide and variable. In moderate and

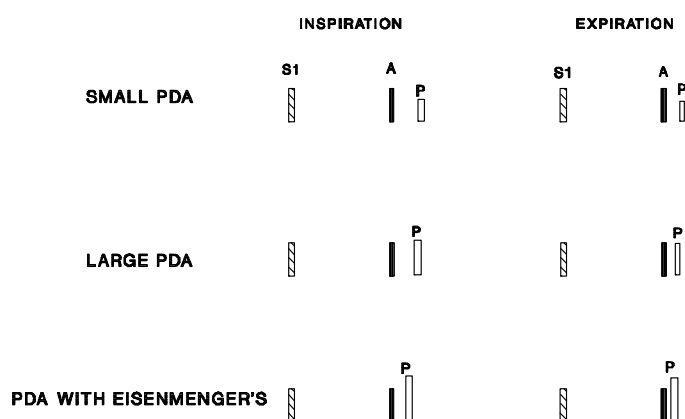


Fig. 18.13: Second sound in patent ductus arteriosus

THE SECOND HEART SOUND

severe pulmonic stenosis, the second heart sound is wide and variably split with a diminished pulmonic sound. The pulmonic sound can be absent in a severe pulmonic stenosis. In the absence of post-stenotic dilatation, the degree of wide split correlates with the severity of pulmonic stenosis (Fig. 18.14). A small ventricular septal defect is often a differential diagnosis for a moderate or severe pulmonic stenosis; the nature of the second heart sound split is helpful in diagnosis. A single second heart sound in a pulmonic stenosis should suggest the possibility of tetralogy of Fallot, dysplastic pulmonary valve or severe pulmonic stenosis itself.

BICUSPID AORTIC VALVE AND AORTIC STENOSIS

In congenital bicuspid aortic valve in the absence of stenosis or regurgitation, the second sound is split normally and the aortic sound is normal or mildly accentuated due to thickening of the valve. In moderate to severe aortic stenosis, the second sound is single or reversed split due to prolongation of left ventricular ejection. When the second heart sound split is reversed in aortic stenosis, the gradient across the aortic valve is usually more than 70 mmHg.

The second heart sound is normal in mild aortic stenosis (Fig. 18.15). The aortic sound may be sometimes accentuated with bicuspid valve. An unusually accentuated aortic sound in a child should raise the suspicion of associated coarctation.

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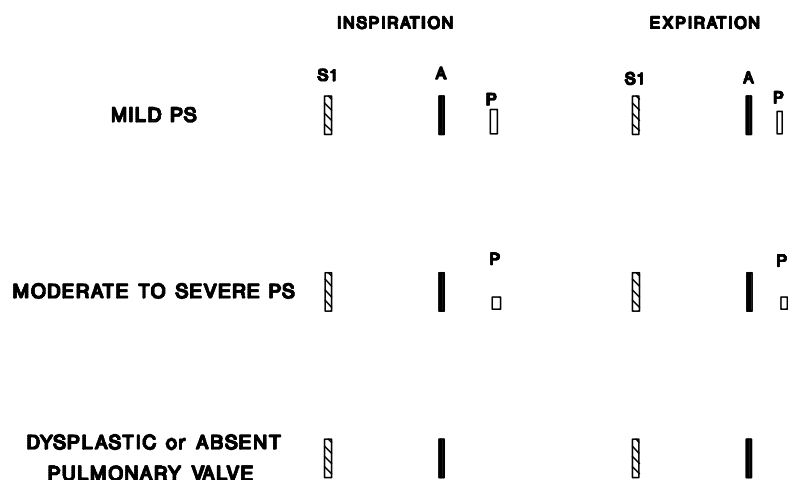


Fig. 18.14: Second sound in pulmonic stenosis of varying severity

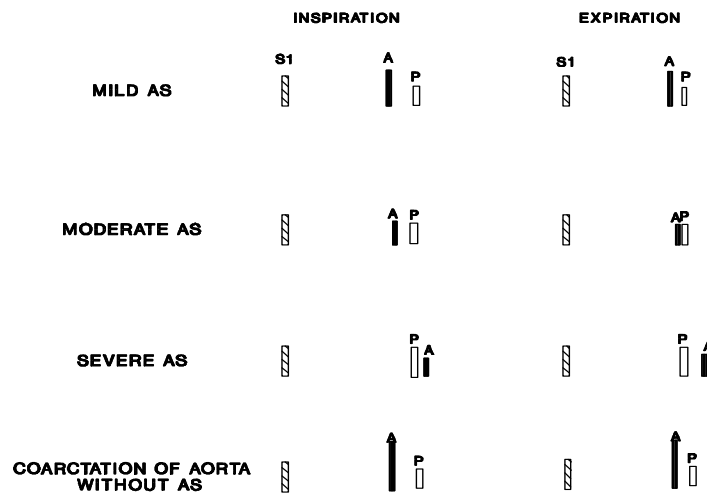


Fig. 18.15: Second heart sound in aortic stenosis (AS)

COARCTATION OF AORTA

The second heart sound is normally split and the aortic sound is accentuated. The accentuation has diagnostic significance in the sense that blood pressures are not routinely recorded in children and the loud aortic sound calls attention to the possible presence of hypertension or coarctation.

Case summary

A two-year-old boy was seen by a pediatrician who suspected a heart murmur and referred him to a cardiologist. After clinical and echocardiographic evaluation, a diagnosis of hypertrophic non-obstructive cardiomyopathy was made and he was referred for further evaluation. An ejection systolic murmur Grade 3/6 was heard along the left sternal border; the ECG showed left ventricular hypertrophy with strain consistent with hypertrophic cardiomyopathy. A repeat echocardiogram was interpreted as consistent with hypertrophic non-obstructive cardiomyopathy. On a review examination the aortic sound was found to be unusually loud for a child and blood pressures were recorded in both upper and lower limbs which was diagnostic of coarctation.

CONGENITAL CYANOTIC HEART DISEASE

The second heart sound is central in the evaluation of cyanotic heart disease. With rare exceptions, it is invariably abnormal in this setting (Table 18.9).

The important features of evaluation are:

Presence or absence of split

- Site of best audibility

THE SECOND HEART SOUND

- If split, normal or wide split

If single

- The site of best audibility
- Intensity

The causes of a wide split second sound in cyanotic heart disease are:

- Total anomalous pulmonary connection
- Single atrium
- Ebstein's anomaly of tricuspid valve
- ASD at fossa ovalis with preferential drainage into left atrium
- ASD with Eisenmenger syndrome
- PS with intact ventricular septum and right to left atrial shunt
- Primary PAH with right ventricular failure and right to left atrial shunt

Table 18.9: Single second heart sound in cyanotic heart disease

<i>Condition</i>	<i>Site of best audibility</i>	<i>Mechanisms</i>
TOF	Single loud A2 LLSB	Absent P2 due to PS Loud A2 due to dilated, dextroposed aorta. Best audibility at LLSB because of normally related aorta
D-TGA	Single loud A2 Left second space	Single S2 due to inaudible P2 due to posteriorly placed PA. Left second space audibility due to superiorly located aorta
L-TGA	Single loud A2 best heard at left second space	Single S2 due to inaudible P2 due to posteriorly placed PA. Left second space audibility
	Split may be audible at right second space	due to superiorly located aorta. Split audible at right second space due to PA placed to the right of aorta
DORV	As in D-TGA	As in D-TGA
Single ventricle	As in D-TGA	As in D-TGA
Tricuspid atresia	Single loud A2 at LSB, apex	Absent P2 due to PS Normally located aortic valve
Truncus arteriosus	Single loud truncal valve closure at LSB, apex	Single because only one semilunar valve present Loud due to dilated truncal root Normally located truncal valve

Table 18.10 Second heart sound in Eisenmenger syndrome

<i>Condition</i>	<i>Nature of split</i>
ASD	Wide and fixed
VSD	Single loud P2
PDA	Close split with normal inspiratory split
VSD of AV canal type	Wide and fixed
TGA, single ventricle, DORV	Single second sound
TAPVC	Wide and fixed

The commonest of cyanotic heart diseases, tetralogy and transposition have a single second heart sound. A normally split second sound is extremely uncommon in cyanotic heart disease and it is a good practice to recheck the physical sign. The causes could be:

- The patient may not be cyanotic
- The second sound may not be split
- If cyanosis is real, consider methemoglobinemia
- Pulmonary arteriovenous fistula
- Vena caval drainage into left atrium

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The Eisenmenger syndrome is often considered the end stage of all left to right shunts. But there is recent evidence to suggest that some forms of this syndrome are still operable with a favourable outcome. Evaluation of the second heart sound in a patient with Eisenmenger syndrome gives a clue to the underlying defect (Table 18.10).

VALVULAR HEART DISEASE

MITRAL STENOSIS

The second heart sound is normally split in mild to moderate mitral stenosis (MS), and is close split or is single in severe mitral stenosis with severe pulmonary hypertension. The intensity of pulmonic sound correlates well with the severity of pulmonary hypertension. The second heart sound opening snap, is often mistaken for a wide split second sound in mitral stenosis. The fact that the first component is louder is a clue that one is dealing with second heart sound and opening snap.

THE SECOND HEART SOUND

In mitral stenosis with pulmonary hypertension, in the combination A2-P2, the last of the two components is louder. When the second sound is wide split in mitral stenosis, an associated atrial septal defect should be considered. Among all valvular lesions, mitral stenosis produces PAH most commonly and consistently. For this reason, if the pulmonic sound is very loud in aortic valve disease, underlying mitral stenosis is likely.

MITRAL REGURGITATION

The second heart sound in mitral regurgitation (MR) gives clues as to the cause, severity and complications of MR. The nature of split is a clue to the severity and cause of mitral regurgitation. The intensity of pulmonic sound is a clue to the complications of MR. The second heart sound in MR when combined with other features gives valuable information in assessment (Table 18.11). One of the common errors is to mistake the third heart sound at the apex as second sound in moderate to severe mitral regurgitation.

AORTIC REGURGITATION

The second heart sound in aortic regurgitation varies depending on the cause, left ventricular function and the associated lesions. The second heart sound is split in the majority of patients with mild, moderate and even severe aortic regurgitation. In severe aortic regurgitation, particularly in association with left ventricular failure the second heart sound may be single or reversed split. The intensity of the aortic sound in aortic regurgitation depends on the cause of the regurgitation. In aortic regurgitation due to aortic root disease, the aortic sound is accentuated; in regurgitation due to valve affection, the aortic sound is diminished or absent. The pulmonic sound can be loud in aortic regurgitation after the onset of left ventricular failure. However, a very loud pulmonic sound in the setting of aortic regurgitation generally means an associated mitral valve disease. Aortic valve disease producing loud aortic sound is unusual but occurs with aortic regurgitation in association with ventricular septal defect and tetralogy of Fallot. Though the list of causes is long for loud aortic sound with aortic regurgitation, they are all uncommon causes for aortic regurgitation. The commonest cause for aortic regurgitation remains rheumatic.

Table 18.11: Second sound in mitral regurgitation

<i>Second sound</i>	<i>MR subset</i>
Normal split	Mild to moderate MR
Wide and variable split	Severe MR Associated RBBB
Wide and fixed split	Severe MR with heart failure Associated ASD MR as part of AV canal defect
Reversed split	MR associated with HOCM Functional MR in severe AS with LVF MR in coronary artery disease Functional MR in cardiomyopathy
Single second heart sound	MR in association with L-TGA Functional MR in severe calcific AS with LVF All causes of reversed split
Normal intensity of P2 with severe MR	The syndrome of severe MR with giant LA with normal LA, PV, and PA pressures
Loud P2 with moderate MR	The subset of less severe MR with smaller LA with high LA, PV and PA pressures

Causes of loud aortic sound in aortic regurgitation (aortic root disease)

Syphilis
 Marfan's syndrome
 Annuloectasia of aortic root
 Rheumatoid arthritis
 Reiter's syndrome
 Takayasu's arteritis
 Aortic dissection
 Functional aortic regurgitation in severe systemic hypertension

Loud A2 with aortic valve disease (prolapse of the leaflet(s))

Ventricular septal defect with aortic regurgitation
 Tetralogy of Fallot with aortic regurgitation

Diminished A2 in aortic regurgitation (aortic valve disease)

Rheumatic heart disease
 In association with aortic stenosis
 Infective endocarditis

THROMBOEMBOLIC PULMONARY ARTERIAL HYPERTENSION

In the earlier stages of the disease, the only evidence is a mild accentuation of pulmonic sound with a normal split. As the disease advances, the pulmonic sound becomes louder and the split is narrower or single. In advanced stages of the disease with right ventricular dysfunction, the split may be wide and fixed. As the disease is asymptomatic in the early stages, and the chances for reversal are higher at this stage, early detection becomes particularly more important. The earliest evidence remains a chance detection of an accentuated pulmonic sound with a narrow split or no split. In general, the pulmonic sound is unimpressive in the early and sometimes even later stages of thromboembolic pulmonary arterial hypertension. Normal intensity of pulmonic sound does not rule out pulmonary hypertension in this setting. For this reason, all patients with recurrent dyspnea, should have a careful echo Doppler evaluation to estimate pulmonary arterial pressures. The threshold to investigate them further by catheterization and angiography should be low.

SECOND SOUND IN A PATIENT WITH SHOCK**333**

If carefully looked for, the second heart sound gives clues to the cause of shock (Fig. 18.16). In non-cardiac causes of shock due to hypovolemia, the second heart

	INSPIRATION			EXPIRATION		
HYPOVOLEMIC SHOCK	S1	A	P	S1	A	P
CARDIOGENIC SHOCK	S1	A P		S1	P A	
RV INFARCTION	S1	A	P	S1	A	P
CARDIAC TAMPONADE	S1	A	P	S1	A	P
ACUTE PULMONARY EMBOLISM	S1	A	P	S1	A	P
TENSION PNEUMOTHORAX	S1	A	P	S1	A	P

Fig. 18.16: Second sound in various shock states

Table 18.12: Second heart sound in shock

<i>Condition producing shock</i>	<i>Second heart sound</i>
Hypovolemic shock	Normal split Pulmonic sound diminished
Cardiogenic shock	Split reversed/normal or single Pulmonic sound accentuated
Right ventricular infarction	Normal split or wide split Pulmonic sound diminished or normal
Cardiac tamponade	Normal split Pulmonic sound normal
Acute pulmonary embolism	Wide split or normal split Pulmonic sound accentuated or normal
Tension pneumothorax	Normal or wide split Pulmonic sound normal or accentuated

sound is normally split and both the aortic sound and pulmonic sound are normal or diminished. In cardiogenic shock as in acute myocardial infarction or myocarditis, the pulmonic sound is accentuated due to the attendant pulmonary hypertension. In shock due to right ventricular infarction, the pulmonic sound is normal or diminished. In pericardial tamponade the pulmonic sound is normal with a normal split. In acute pulmonary embolism, the second heart sound may be wide split with a loud pulmonic sound. However, in many patients, the pulmonic sound may not be loud due to acute severe right ventricular dysfunction (Table 18.12).

Thus the evaluation of second sound is of immense value in the assessment of the majority of heart lesions. With a normal split second heart sound and normal intensity of both components, no significant heart disease is likely to be present. Even if heart disease exists with a normal second heart sound, there is no precipitate need for invasive investigation or surgical intervention. For that matter if the second heart sound is normal in split with normal intensity of both components, congenital heart disease is unlikely.

19 The Third Heart Sound

The third heart sound, also called a ventricular gallop or protodiastolic gallop, follows the second heart sound during rapid ventricular filling.

MECHANISMS

Though various mechanisms are proposed for the genesis of third sound (Table 19.1, Fig. 19.1), the most consistent feature remains rapid ventricular filling. The proposed mechanisms are valvular, ventricular and the impact of the left ventricle on the chest wall. More than one mechanism may be responsible.

Table 19.1: Mechanisms of third heart sound

Valvular
Ventricular
Left ventricular chest wall interaction
Sudden limitation in the long axis filling
Movement of the ventricle

With the opening of the mitral valve, there is a sudden rush of blood from the left atrium to the left ventricle, which is halted abruptly in situations where early compliance of the LV chamber is decreased. Thus, S3 corresponds to the peak of rapid filling wave (RF) seen in an apexcardiogram. The normal A2-S3 interval in LV varies between 120 and 160 msec. In pathological states, the third heart sound is of early onset and is loud.

Whatever the mechanism, a sudden inherent limitation in the long axis filling movement of the left ventricle is consistently observed. The third heart sound occurs in all situations where there is rapid filling of the ventricle, and can happen

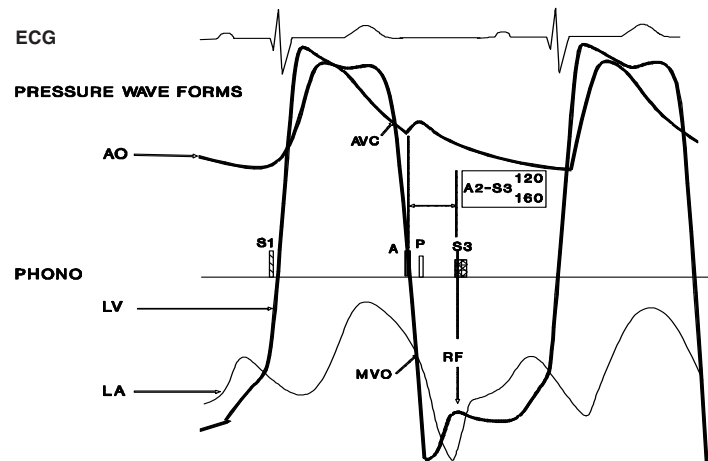


Fig. 19.1: Mechanism of third heart sound (S3).

S1: first heart sound, A2: aortic closure sound, P2: pulmonic closure sound, S3: third heart sound,
MVO: mitral valve opening, AVC: aortic valve closure, RF: rapid filling of ventricle

on either side of the heart. In the presence of AV valve stenosis no third heart sound is possible, as rapid filling of the ventricle is impossible in this setting.

Table 19.2: Causes of third heart sound

Category	Causes
Physiological	Children Young adults (< 40 years) Pregnancy
Pathological	Ventricular failure (RV or LV) <i>Non-heart failure causes</i> Hyperkinetic circulatory state Anemia Thyrotoxicosis Beri-beri Volume overload AV valve regurgitation MR/TR Semilunar valve regurgitation AR/PR Systemic arteriovenous fistula Left to right shunts ASD/VSD/PDA
S3 like sounds	Pericardial knock Tumour sound as in left atrial myxoma

THE THIRD HEART SOUND

Conditions producing the third heart sound can be grouped as physiological and pathological. Pathological states could further be classified as heart failure and non-heart failure states. In children and young adults, the third heart sound is normally heard. It is not heard in normal infants and in adults beyond 40 years of age. There are no distinctive features by which one can differentiate a physiological from a pathological third heart sound. However, cardiac enlargement, in association with loudness and early onset of third heart sound, generally signify a pathological cause.

Though ventricular failure is the most important cause of third heart sound, there are many other causes of third heart sound (Table 19.2). In all these states the presence of the third heart sound should not be used as evidence of heart failure. An easily audible third heart sound is less common with aortic regurgitation than mitral regurgitation of similar degree. In fact, a clearly audible third heart sound in the setting of aortic regurgitation generally suggests heart failure.

The pericardial knock of constrictive pericarditis for all practical purposes is not distinguishable from the third heart sound. It is usually sharper, earlier in onset, best heard along the left sternal border and increases during inspiration. The tumor sound of left or right atrial myxoma can simulate a third heart sound. A sound that is similar to the third heart sound in the presence of mitral stenosis with little or no mitral regurgitation, may give a clue to left atrial myxoma.

In certain clinical states, the third heart sound occurs early and is close to the preceding second heart sound. The range of second heart sound to third heart sound interval at ordinary heart rates is 0.12–0.16 seconds. An early third heart sound with normal heart rates of 70–90/minute is considered to be a feature of constrictive pericarditis. Apart from constriction, any condition with very high

Table 19.3: Causes and mechanisms of third heart sound

<i>Causes of early third heart sound</i>	<i>Mechanisms of early third heart sound</i>
Constrictive pericarditis Severe MR with large V wave Severe TR with large V wave Acute severe AR	High venous or atrial pressures Very rapid reversal of pressures during early diastole Very large V wave as in severe MR with small left atrium
Any severe ventricular failure	Small ventricular cavity with high filling pressures as in constriction

venous or atrial pressures and quick reversal of pressures in early diastole may have an early third heart sound.

The large V wave of severe mitral regurgitation is responsible for the early third heart sound seen in this condition. Any severe ventricular failure with extreme elevation of venous pressures behind the failing ventricle may cause an early third heart sound. In a patient with features of congestive heart failure, a third heart sound is almost always audible. An absent third heart sound in the presence of 'congestive heart failure' may suggest the possibility of pericardial tamponade or AV valve stenosis. Otherwise the diagnosis of heart failure should be rechecked.

Correlations

In the presence of heart failure, the third heart sound correlates well with the ventricular end diastolic pressures and is usually above 25 mmHg on the left side. The right sided third heart sound correlates with a rapid γ descent in the neck veins. Other correlations are shown in Table 19.4.

AUSCULTATION TECHNIQUE

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The third heart sound is a low frequency sound best heard with the bell; it can occur on either side of the heart (Table 19.5). The left ventricular third heart sound is best heard at the apex with the bell in the left lateral position during expiration and is accentuated by isometric hand grip. Unless it is very loud, it is localized to the apex. The right ventricular third heart sound on the other hand is best heard along the lower left sternal border, increases during inspiration and passive leg raising in supine position. It decreases in standing position and during expiration. As the human ear is least efficient in appreciating low frequency sounds in the range of third heart sound and fourth heart sound, we must carefully check

Table 19.4: Correlates of third heart sound

<i>Anatomical</i>	Dilated ventricle
<i>Functional</i>	Systolic dysfunction (EF < 40%)
<i>Hemodynamic</i>	
LVEDP	> 25 mmHg
Cardiac index	< 2 l/min/m ²
Symptoms	Dyspnea, PND, orthopnea
Doppler flow across AV valves	Tall E wave compared to A wave

THE THIRD HEART SOUND

Table 19.5: Features of left ventricular versus right ventricular third heart sound

<i>Feature</i>	<i>LV S3</i>	<i>RV S3</i>
Site	Apex	LLSB
Respiration	Better heard during expiration	Inspiration
Position	Left lateral	Supine position Passive leg raising
Isometric hand grip	Increases	No change
Associated features	Left sided cause for S3	Elevated JVP Rapid γ descent

for these sounds when the clinical circumstance demands. One simple method is to start hearing at the pulmonic area and concentrate in the interval following the second heart sound. Normally, this period in diastole is impressively silent at the pulmonary area. After appreciating this silence following the second heart sound, any extra sound appearing immediately after the second heart sound as the stethoscope is moved toward apex, is easy to make out. Other sounds can be heard using a similar method.

Once third heart sound is detected, one must mention whether it is right ventricular or left ventricular, whether it is early or late, and whether it is physiological or pathological.

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SIGNIFICANCE OF THIRD HEART SOUND IN VARIOUS DISORDERS

Normal person

A physiological third heart sound can be heard in practically all healthy children and adolescents but rarely in people beyond 40 years. This is attributed to vibrations of the heart muscle arising from prominent early diastolic left ventricular filling and the subsequent abrupt inflow deceleration. The reason why it fades with age is unknown. The prevalence of physiological S3 decreases in parallel with the reduction of the early diastolic left ventricular filling rate. The primary cause is thought to be an age related increase in blood pressure with the development of relative left ventricular hypertrophy and altered diastolic compliance. Kupari et al in a population based study showed that nearly 25 per cent of healthy persons aged 36–37 years have an audible physiological S3. The presence of this sound is

associated independently with leanness and with a relatively high peak early diastolic transmitral velocity. With advancing age, the third sound disappears as the left ventricular early filling decreases. The basic mechanism is probably not left ventricular hypertrophy caused by increased blood pressure, as is widely believed but a more primary alteration (ageing) of the left ventricular diastolic properties.

The third heart sound in normal children and during pregnancy can be confused with mitral or tricuspid diastolic murmurs. The absence of any abnormality of second sound, or absence of cardiac enlargement, are helpful signs in diagnosis.

Mitral regurgitation

In adults with mitral regurgitation, the third sound helps in assessment of severity of mitral regurgitation. Any mitral regurgitation beyond mild mitral regurgitation is generally accompanied by the third heart sound. Moderate or severe mitral regurgitation is almost always accompanied by the third heart sound. In a diagnosis of moderate or severe mitral regurgitation, if the third heart sound is not heard one must suspect an aortic stenosis simulating mitral regurgitation. As the third heart sound is normally heard in children and adolescents, it cannot be used to estimate the severity of mitral regurgitation. In severe mitral regurgitation, the loudest sound at the apex is often the third heart sound and is often confused for the second heart sound. An apical mid-diastolic murmur is common with severe mitral regurgitation and the accompanying third sound rules out an associated mitral stenosis. The third heart sound is so consistently present in mitral regurgitation, that when a lesion, which is generally not accompanied by a third heart sound, is diagnosed, the presence of the third heart sound makes associated mitral regurgitation likely, as in aortic stenosis with mitral regurgitation.

Aortic regurgitation

Though the third heart sound is expected to occur in aortic regurgitation as in mitral regurgitation, in actual practice *third heart sound is rare in aortic regurgitation* in the absence of left ventricular failure. An easily audible third heart sound in aortic regurgitation generally means a complicating left ventricular failure or an associated mitral regurgitation. Aortic regurgitation can produce a third heart sound even in the presence of associated mitral stenosis as rapid filling can occur through the aortic valve. In acute aortic regurgitation, the first heart sound is absent or

diminished due to premature closure of the mitral valve and the only sound at the apex is a loud third heart sound. This third heart sound is often mistaken for the first heart sound and the short early diastolic murmur of aortic regurgitation occurring along with third heart sound is mistaken for a systolic murmur. In the setting of unexplained acute left ventricular failure or pulmonary edema one must look for this combination of auscultatory findings. The mistake is compounded by the fact that acute aortic regurgitation is not accompanied by the peripheral arterial signs of aortic regurgitation, due to a high left ventricular end-diastolic pressure.

Myocardial disease

In myocardial disease, the third heart sound provides insights into the anatomical and functional nature of the disorder and allows a classification of the disease as congestive or restrictive types. The third heart sound in this setting reflects systolic dysfunction and the ejection fraction is usually less than 40 per cent. In restrictive cardiomyopathies, the third heart sound occurs only when frank congestive failure develops.

Congenital cyanotic heart disease

The third heart sound is not heard in tetralogy of Fallot, which is the commonest cyanotic heart disease in children beyond 2 years of age. It is also not a feature of lesions with a tetralogy like physiology, namely, a ventricular septal defect with pulmonary stenosis. But the third heart sound is a feature of all cyanotic heart diseases with increased pulmonary blood flow as in total anomalous pulmonary venous connection, double outlet right ventricle without pulmonic stenosis, truncus arteriosus and D-transposition of great arteries with ventricular septal defect. The third heart sound also occurs in some cyanotic disorders with decreased pulmonary flow as in Ebstein's anomaly of tricuspid valve or severe pulmonic stenosis with intact ventricular septum and right to left atrial shunt with heart failure.

Heart failure

A third heart sound is almost always heard in ventricular failure. This is more often true in chronic disorders. In a significant number of patients with acute myocardial infarction, the third heart sound is not easily audible in spite of left ventricular failure. In this setting, the respiratory rate is a better indicator of LVF than the third heart sound. In the presence of 'heart failure', if the third heart

sound is not heard, one must consider the possibility of cardiac tamponade or mitral or tricuspid stenosis.

The causes of heart failure without third heart sound are:

- Pericardial tamponade
- Mitral valve obstruction
- Tricuspid valve obstruction
- Acute myocardial infarction with left ventricular failure in some patients

Shock syndrome

In a patient with shock or hypotension, presence of the third heart sound distinguishes the cardiogenic cause of shock from the non-cardiogenic cause. In the absence of the third heart sound any of the non-cardiogenic causes of shock like hypovolemia or septic shock should be considered. Absence of a third heart sound is a feature of shock due to cardiac tamponade and tension pneumothorax.

The causes of shock without third heart sound are:

- Hypovolemic shock
- Septic shock
- Cardiac tamponade
- Tension pneumothorax

Atrial septal defect

In an atrial septal defect the expected *right ventricular third heart sound is not heard* and the tricuspid diastolic murmur is a more consistent finding. The combination of normal variant innocent systolic murmur and audible expiratory split is often mistaken for an atrial septal defect in children. The physiological left ventricular third sound in children often excludes an atrial septal defect as the left ventricle is under filled in atrial septal defect.

A third heart sound in 'atrial septal defect' should suggest the possibility of Ebstein's anomaly mistaken for atrial septal defect. Other possibilities are, atrial septal defect with mitral valve disease, and ostium primum atrial septal defect with mitral or tricuspid regurgitation. In the absence of any of the above conditions, right ventricular cardiomyopathy due to volume load should be considered.

20 The Fourth Heart Sound

MECHANISM

The fourth heart sound is also called the atrial or pre-systolic gallop. It precedes the first heart sound and is a low frequency sound, like the third heart sound. The fourth heart sound arises in the ventricle due to a forcible atrial contraction against a non-compliant ventricle and can arise on either side of the heart. The requisites for a fourth heart sound are a healthy atrium in sinus rhythm, a non-stenotic AV valve and a non-compliant non-dilated ventricle.

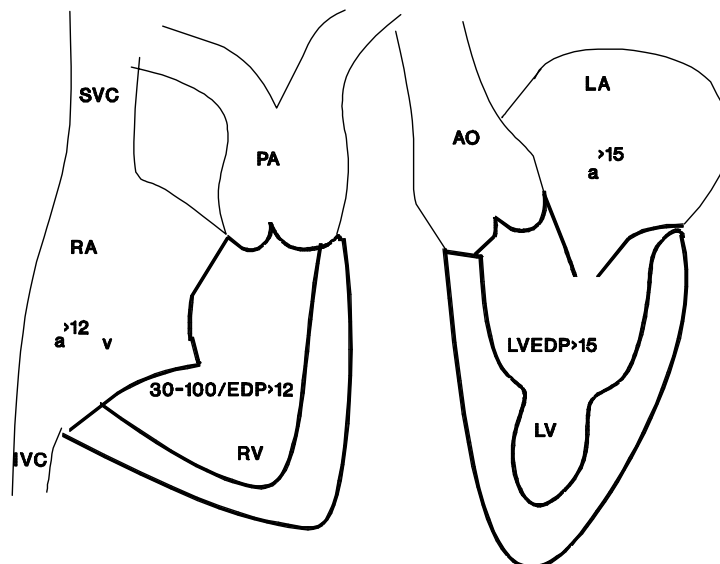


Fig. 20.1: Pressure correlations of fourth heart sound (S₄). For generation of fourth heart sound RVEDP has to be more than 12 mmHg and LVEDP has to be more than 15 mmHg.

The fourth heart sound best correlates with ventricular end-diastolic pressure (EDP) and height of *a* wave in atrial pressure tracing (Fig 20.1). The fourth heart sound can be heard when right ventricular EDP is > 12 mmHg on the right side, and left ventricular EDP > 15 mmHg on the left side. If the EDP is very high, that is, greater than 25 mmHg, the fourth heart sound may be absent because of insufficient pumping function of atria.

The requisite factors for a fourth heart sound are:

1. Atrial factors
A healthy atrium
2. Atrioventricular valve factors
Non-stenotic AV valve
3. Ventricular factors
Non-compliant non-dilated ventricle as in
 - Ischemia
 - Infarction
 - Concentric hypertrophy
 - Restrictive myopathy
 - Acute volume load
4. Rhythm: sinus rhythm

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When the atrial and ventricular factors are favourable, ventricular non-compliance is the basis for the fourth heart sound. In a person beyond 60 years of age, a fourth sound is usually recordable in phonocardiograms. However an easily audible fourth heart sound at any age is generally abnormal.

Causes

Physiological (recordable not audible)

- Elderly people age above 60 years

Pathological

All causes of concentric left ventricular hypertrophy:

- Systemic hypertension (moderate/severe)
- Aortic stenosis (moderate/severe)
- Hypertrophic cardiomyopathy (obstructive/non-obstructive)
- Restrictive cardiomyopathies

THE FOURTH HEART SOUND

All causes of concentric right ventricular hypertrophy:

- Pulmonic stenosis (moderate/severe)
- Pulmonary arterial hypertension (moderate/severe)
- Restrictive cardiomyopathy

Coronary artery disease:

- Ischemia or infarction

Acute regurgitant lesions:

- Acute aortic regurgitation
- Acute mitral regurgitation
- Acute tricuspid regurgitation
- Prolonged P-R interval

CLINICAL RECOGNITION

The fourth heart sound is a low frequency sound and is capable of arising on either side of the heart. The left ventricular fourth heart sound is best heard at the apex with the bell of the stethoscope lightly applied during expiration. In contrast to the third heart sound, the presence of which may mean ventricular failure, the fourth heart sound does not indicate heart failure. It only signifies a 'hardworking ventricle'. Of late the importance of diastolic function of the ventricle is increasingly being realized. Isometric hand grip accentuates it. The right ventricular fourth heart sound is best heard at the lower left sternal border with the bell, in supine position. It increases during inspiration and passive leg raising. Unlike its left sided counterpart the right sided fourth heart sound is more widely audible along the left sternal border and in the jugular notch.

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SIGNIFICANCE IN VARIOUS DISORDERS

The fourth heart sound correlates with a hypertrophied non-dilated ventricle; the left ventricular end diastolic pressure is usually above 15 mmHg (Table 20.1).

Left ventricular outflow obstruction

All forms of left ventricular outflow obstruction of more than moderate degree generally result in concentric LVH with reduction in ventricular compliance. The presence of the fourth heart sound correlates with a gradient of at least 50 mmHg across the left ventricular outflow. This correlation with the degree of obstruction is not applicable in hypertrophic cardiomyopathy where the fourth heart sound is

Table 20.1: Correlates of fourth heart sound

Anatomical	Concentric ventricular hypertrophy with small cavity Non-hypertrophied Ischemia Infarction Restrictive cardiomyopathy
Functional	Normal or supernormal systolic function EF > 50% Reduction in end-diastolic volume
Hemodynamic	LVEDP > 15 mmHg
Electrocardiographic	Normal sinus rhythm P-R interval > 0.14 sec
Echo Doppler	Ventricular hypertrophy of pressure loaded pattern Atrial enlargement Prominent <i>a</i> wave Increase in wall thickness to cavity ratio Exaggerated late diastolic wave pattern in AV valve flow
Clinical	
JVP	Prominent <i>a</i> wave
Cardiac impulse	Pre-systolic impulse

consistently present irrespective of the presence or the degree of obstruction. With associated coronary artery disease or hypertension, a milder obstruction may have a fourth heart sound. In the presence of severe aortic stenosis associated mitral stenosis may be masked due to obliteration of the pressure gradient across the aortic valve. The presence of the fourth heart sound in this setting rules out mitral stenosis. The fourth heart sound of aortic stenosis is often better palpable than audible due to the accompanying loud systolic murmur of aortic stenosis masking the preceding fainter sound.

Even with severe aortic stenosis, the fourth heart sound may not be audible in the presence of associated mitral stenosis or a complicating atrial fibrillation.

Right ventricular outflow obstruction

The fourth heart sound occurs when the obstruction is at least moderate. The RV pressure is usually in the systemic range when a fourth heart sound is heard. The right ventricular fourth heart sound correlates with a prominent *a* wave in the jugular venous pulse measuring more than 15 cm and the right ventricular

end diastolic pressure is usually more than 12 mmHg. The right ventricular fourth heart sound is more widely audible than its counterpart on the left side. It is heard along the left sternal border and frequently over the jugular notch. Occasionally, the fourth heart sound in severe pulmonic stenosis may be relatively long and may simulate a pre-systolic murmur of tricuspid stenosis. The fourth heart sound of pulmonic stenosis is better heard during inspiration and supine position or leg raising. The inspiratory increase in fourth heart sound is accompanied by a simultaneous decrease or disappearance of the ejection click of pulmonic stenosis. This is related to the premature opening of the pulmonic valve due to the right ventricular end diastolic pressures exceeding pulmonary artery diastolic pressures. The fourth heart sound of pulmonic stenosis is also an indication of an intact ventricular septum, and generally of the atrial septum too. In the presence of associated ASD or VSD the right atrial force is dissipated into the left atrium or the left ventricle respectively.

Mitral regurgitation

The fourth heart sound is not a feature of chronic mitral regurgitation as ventricular dilatation is common in this setting. However, the fourth sound occurs in mitral regurgitation when it is acute or chronic mitral regurgitation with ventricular non-compliance.

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Causes

- Acute mitral regurgitation
 - Infective endocarditis
 - Papillary muscle dysfunction or rupture
 - Chordal rupture
 - Post closed or open commissurotomy
 - Post balloon dilatation of mitral valve for mitral stenosis
 - Spontaneous as in mitral valve prolapse
 - Trauma
 - Prosthetic malfunction
- Chronic mitral regurgitation
 - Coronary artery disease with papillary muscle dysfunction
 - Hypertrophic obstructive cardiomyopathy
 - Endomyocardial fibrosis

Table 20.2: Mechanisms for absence of fourth heart sound in rheumatic mitral regurgitation

<i>Feature</i>	<i>Mechanism</i>
Chronic mitral regurgitation	Dilated compliant left ventricle
Associated mitral stenosis	Force of atrial contraction cannot be transmitted to left ventricle
Atrial fibrillation	Absence of atrial contraction
Dilated unhealthy left atrium	Atrium incapable of generating enough force

In the commonest form of mitral regurgitations, namely rheumatic, the fourth heart sound is consistently absent (Table 20.2). The mechanisms precluding the fourth heart sound in rheumatic mitral regurgitation are, the chronicity of mitral regurgitation with a dilated left ventricle, atrial fibrillation, associated mitral stenosis and a dilated, unhealthy left atrium.

Aortic regurgitation

Chronic aortic regurgitation is not usually accompanied by the fourth heart sound. The fourth sound occurs when aortic regurgitation is acute or when the ventricular compliance is reduced by an associated disorder. When a fourth sound is heard in aortic regurgitation, an associated mitral stenosis can be ruled out with confidence but a third heart sound is possible in spite of mitral stenosis as the ventricle can be rapidly filled from the aorta.

Causes

- *Acute aortic regurgitation*
 - Infective endocarditis
 - Post-balloon dilatation for aortic stenosis
 - Post-surgical valvotomy
 - Prosthetic malfunction
 - Dissecting hematoma of ascending aorta
 - Retroversion of aortic cusp as in aortic root disease
- *Chronic aortic regurgitation*
 - Functional aortic regurgitation with severe systemic hypertension
 - Severe aortic stenosis with milder aortic regurgitation
 - Milder aortic regurgitation with associated coronary artery disease

THE FOURTH HEART SOUND

Myocardial disease

The fourth heart sound in this setting allows the distinction between dilated and restrictive cardiomyopathies. In the dilated form, the fourth heart sound is obviously not a feature. The most important sound in restrictive cardiomyopathy is the fourth heart sound. It is a reflection of a well maintained systolic function or ejection fraction and an impaired diastolic function or relaxation of the ventricle.

Coronary artery disease

Clearly audible and palpable fourth heart sound in coronary artery disease is common. It usually denotes diastolic dysfunction with left ventricular end diastolic pressure more than 18 mmHg. The most common cause of diastolic dysfunction in this setting is myocardial ischemia or myocardial infarction. However, one should also realize that hypertension which is commonly seen with coronary artery disease can also generate a fourth heart sound. Additionally, patients with aortic stenosis, either fixed or dynamic, can also have angina, fourth heart sound. These patients usually have left ventricular hypertrophy and diastolic dysfunction. In the setting of myocardial infarction, an audible fourth heart sound indicates that at least 10 per cent of myocardium is at jeopardy. In a patient with shock, a fourth heart sound indicates that hypovolemia is unlikely as wedge pressure will be more than 18 mmHg.

21 Ejection and Non-ejection Clicks

EJECTION CLICKS

Clicks can be ejection or non-ejection. Ejection clicks in turn can be valvular or vascular in origin. Normally, the semilunar valves open noiselessly. When the aortic or pulmonic valve open noisily, ejection clicks occur. A valvular ejection click occurs due to stenosis or deformity of either semilunar valve and is due to an abrupt doming motion of the valve coming to an abrupt halt. The doming and halting motion seen on echocardiogram coincides with the clinically audible click.

Clicks can result either from abnormality of the valve or the root of the major vessels (aorta and pulmonary artery). In valvular stenosis, the click results

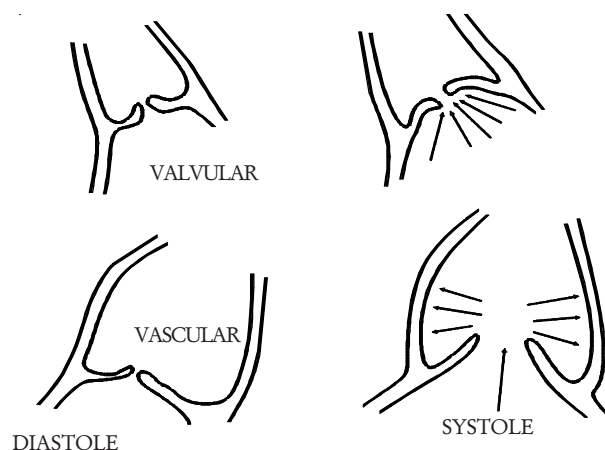


Fig. 21.1: Mechanism of ejection click (EC)

EJECTION AND NON-EJECTION CLICKS

from abrupt halting of a doming valve at the onset of ejection. The post-stenotic dilatation contributes in the genesis of ejection click to some extent. Sometimes, an ejection click can result solely from dilatation of the root due to halting motion of the compliant root at the time of ejection.

VALVULAR CLICKS

Mechanism

The click is caused by the doming and abrupt halting movement of the valve.

Causes

- Aortic valve
 - Valvular aortic stenosis
 - Congenital bicuspid aortic valve
 - Congenital quadricuspid 'aortic valve' as in truncus arteriosus
- Pulmonary
 - Valvular pulmonary stenosis

VASCULAR CLICKS

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Vascular clicks on the other hand arise due to sudden distension of the vessel beyond the valve along with the opening movement of the valve. The mechanism and significance, and the causes are given in Tables 21.1 and 21.2.

Table 21.1: Mechanism and significance of vascular clicks

<i>Mechanism</i>	<i>Significance</i>
Increased pressure beyond the valve	Systemic hypertension Pulmonary hypertension
Increased flow across the valve	Hyperkinetic circulatory states Left to right shunts for pulmonary side Regurgitation of semilunar valves
Dilatation of the vessel beyond the valve	Dilatation or aneurysm of ascending aorta Dilatation of pulmonary artery due to <ul style="list-style-type: none">Increased flowIncreased pressureIdiopathic dilatationCombination of the above factors

Table 21.2: Causes of aortic and pulmonary vascular clicks

<i>Aortic vascular clicks</i>	<i>Pulmonary vascular clicks</i>
Systemic hypertension	Pulmonary arterial hypertension
Aneurysm of ascending aorta	Left to right shunts
Aortic regurgitation	Anemia
Tetralogy of Fallot	Thyrotoxicosis
Anemia	Idiopathic dilatation of pulmonary artery
Thyrotoxicosis	

Table 21.3: Vascular versus valvular clicks

<i>Feature</i>	<i>Vascular click</i>	<i>Valvular click</i>
<i>Second heart sound</i>		
Intensity	Loud	Diminished or normal
Split	Normal or close split or single S2	Abnormal split wide or reversed
<i>Palpable artery beyond the valve</i>	Often palpable particularly pulmonary artery	Impalpable

The corresponding second heart sound is generally loud in vascular clicks and is diminished or normal in valvular clicks. Though the aortic sound and pulmonary sound are often described as diminished or absent in aortic stenosis and pulmonic stenosis, in mild lesions the corresponding second heart sound is often normal or even mildly increased. This is related to thickened and mobile aortic or pulmonary valves. For similar reasons, the aortic sound in a bicuspid aortic valve is often sharper than normal.

EJECTION CLICK OF AORTIC STENOSIS

The ejection click of valvular aortic stenosis is usually best heard at the left second space or aortic area and is also widely audible along the left sternal border and apex. Occasionally it is heard best at the apex in which case it can be mistaken for a loud first heart sound or a split first heart sound.

It is classically heard in aortic stenosis of bicuspid valve. The intensity correlates with the pliability of the valve. It occurs earlier as the severity of aortic stenosis increases and corresponds to the anacrotic wave.

EJECTION AND NON-EJECTION CLICKS

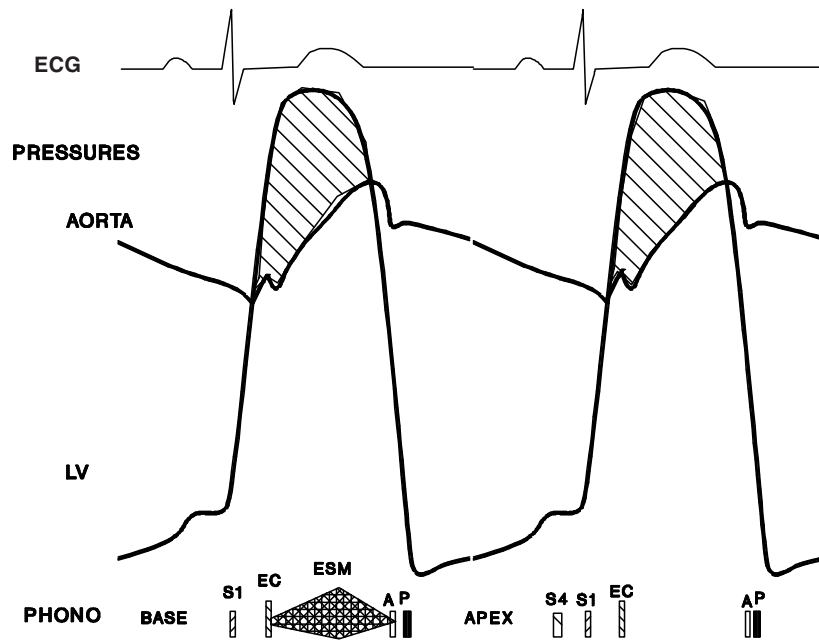


Fig. 21.2: Aortic ejection click

Significance

- Best audible at the aortic area but is widely audible
- Constant
- Localizes the obstruction to the valve
- May be absent when the valve is calcified or immobile
- Is often mistaken for a loud first heart sound
- Absence of ejection click in a child with aortic stenosis makes valvular aortic stenosis unlikely; sub valvular aortic stenosis or some other diagnosis is likely

The ejection click of valvular aortic stenosis is constant and does not vary with respiration unlike that of valvular pulmonic stenosis. Unlike in pulmonic stenosis, a premature opening of the aortic valve never occurs in aortic stenosis. (The left ventricular end diastolic pressure is never higher than aortic diastolic pressure). As the ejection click is often clearly heard at the apex, it can be mistaken for a loud S1. The ejection click is best heard at the base unlike the first heart sound, which is best heard at the apex. In children with valvular aortic stenosis the ejection click is always heard but can be absent in adults with calcified valves.

If an ejection click is not heard in a child with aortic stenosis, either aortic stenosis is unlikely or there is an obstruction somewhere else (such as sub valvular or supra valvular aortic stenosis). Calcification of the aortic valve is a rule in aortic stenosis beyond the age of forty, particularly in males. Persistence of aortic valvular ejection click in an elderly person with aortic stenosis usually indicates a milder obstruction.

Ejection click of congenital bicuspid aortic valve

A bicuspid aortic valve is one of the commonest congenital anomalies, occurring in 2 per cent of all live births. Its importance lies in its predisposition to infective endocarditis and the potential development of aortic stenosis or regurgitation or both.

A loud aortic ejection click may be the only manifestation in the absence of aortic stenosis or aortic regurgitation (Fig. 21.3). In the absence of accompanying aortic stenosis or aortic regurgitation, the click is often mistaken for a loud first heart sound. However, whenever the first heart sound is louder at the base than at the apex, an ejection click is likely. In all patients presenting with fever one must look for this sign, as it is important to diagnose infective endocarditis early.

The combination of a loud ejection click heard well at the apex resembling a loud S₁, and an accompanying early diastolic murmur of aortic regurgitation heard clearly at the apex, often leads to a mistaken diagnosis of mitral stenosis. However, the 'loud S₁', louder at the base than at the apex, and what sounds like a widely audible S₁ split, are clues to aortic regurgitation due to bicuspid aortic valve (Fig. 21.4).

EJECTION CLICK OF VALVULAR PULMONARY STENOSIS

The ejection click is best heard in the pulmonary area and is usually localized to that area. In moderate to severe pulmonic stenosis, the click varies with respiration

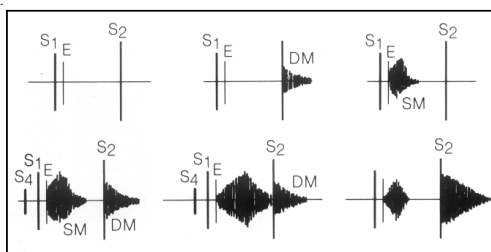


Fig. 21.3: Spectrum of auscultatory findings in bicuspid aortic valve

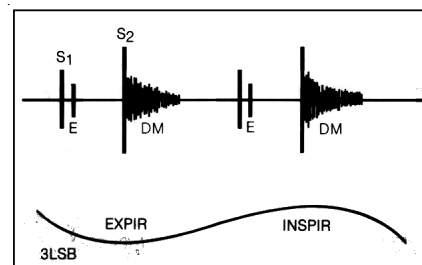


Fig. 21.4: Congenital bicuspid aortic valve with a loud ejection click and aortic regurgitation

and is better heard or audible only during expiration but is diminished or absent during inspiration. In extremely severe pulmonic stenosis, the click may be heard only during expiration or may be absent altogether. The variability of the click is related to the elevated right ventricular end diastolic pressure (RVEDP) in moderate or severe pulmonic stenosis due to concentric right ventricular hypertrophy (Fig. 21.5). During inspiration, the increased venous return to the right ventricle leads to elevation of RVEDP beyond the pulmonary artery diastolic pressure resulting in premature opening of the pulmonary valve in diastole itself. In milder degrees of pulmonic stenosis, as the RVEDP is normal, premature opening of the pulmonic valve is not possible and the click may not vary.

Normally, with inspiration the pulmonary arterial diastolic pressure falls and right ventricular diastolic pressure may remain unchanged as venous return increases or may actually fall due to transmission of negative intrapleural pressure. However, in valvular pulmonic stenosis, as the right ventricle is hypertrophied, the right ventricular diastolic pressure tends to rise as venous return increases whereas

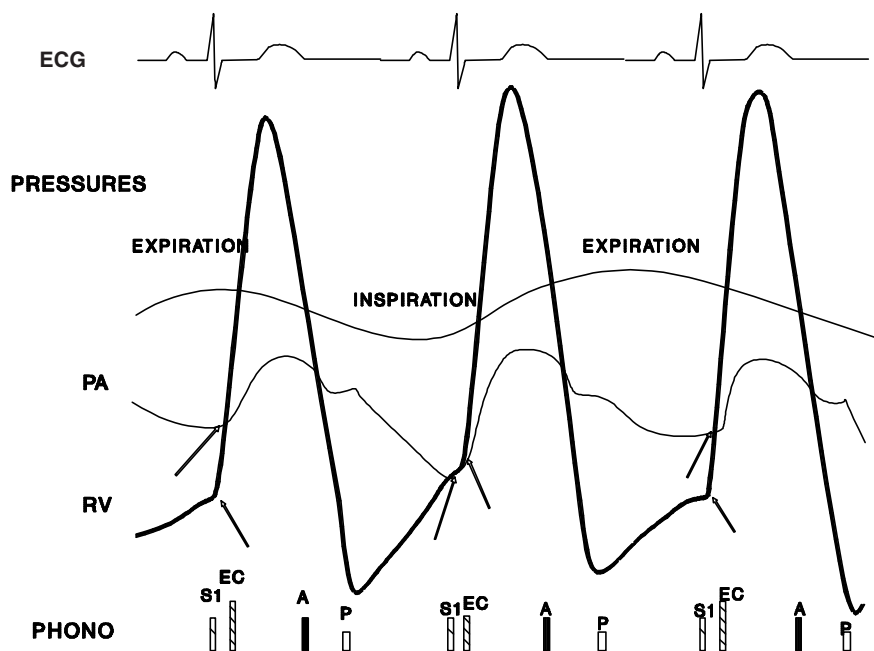


Fig. 21.5: Mechanism of variable ejection click in valvular pulmonic stenosis

pulmonary arterial pressure falls (as expected). This induces premature opening of the pulmonary valve before the onset of systole due to elevation of right ventricular diastolic pressure that exceeds pulmonary diastolic pressure. This premature opening decreases the intensity of the ejection click in inspiration (Fig. 21.6).

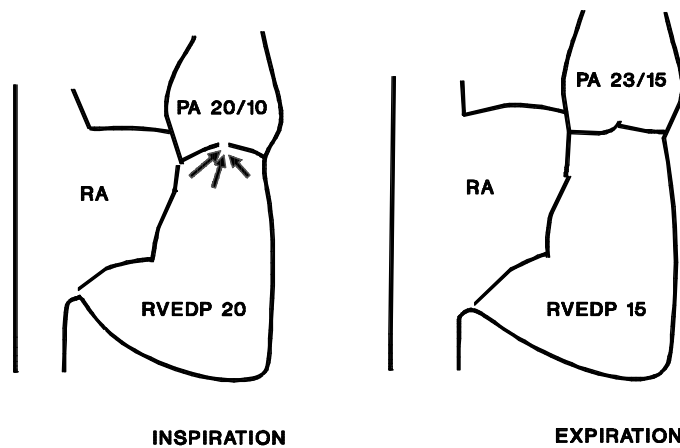


Fig. 21.6: Influence of respiration on pulmonary ejection click in pulmonary stenosis

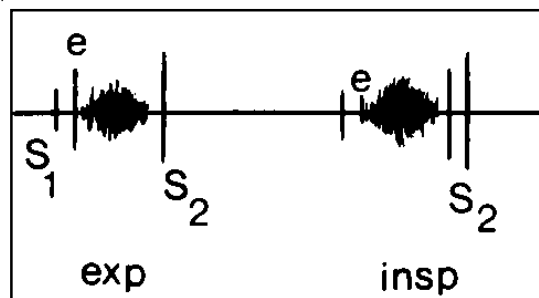
	INSPIRATION			EXPIRATION		
	S1EC	A	P	SEC	A	P
PULMONARY STENOSIS						
	S1EC	AP		S1EC	PA	
AORTIC STENOSIS						

Fig. 21.7: The ejection click and second heart sound in valvular aortic and pulmonic stenosis. The ejection click in pulmonic stenosis varies in intensity with respiratory phases (increasing in expiration and decreasing in inspiration), whereas that of aortic stenosis does not.

EJECTION AND NON-EJECTION CLICKS

Table 21.4: Aortic versus pulmonary ejection clicks

<i>Feature</i>	<i>Aortic click</i>	<i>Pulmonary click</i>
Site of best audibility	Aortic area	Pulmonary area
Conduction	Widely audible	Localized to pulmonary area
Relation to respiration	Constant	Variable
Accompanying features	LVH	Better heard during expiration
	Slow rising carotid pulse	RVH
	Systolic thrill at aortic area, neck	Prominent <i>a</i> wave in JVP
	Reversed split, diminished or absent A2	Systolic thrill at pulmonic area
		Wide split S2 with diminished P2



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Fig. 21.8: Expiratory increase of ejection click of pulmonic stenosis

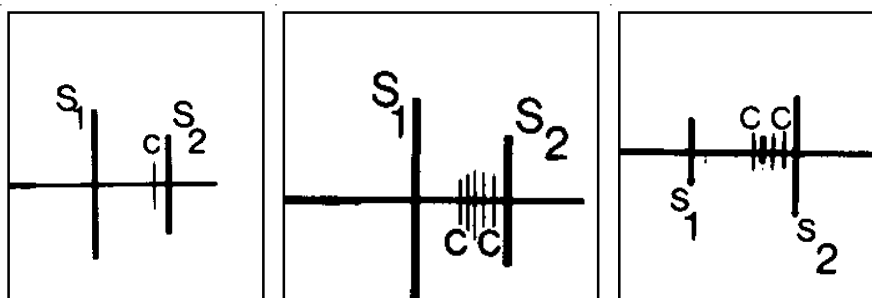


Fig. 21.9: Clicks in mitral valve prolapse may be multiple and vary in timing

Similarly, the second heart sound split is very wide in pulmonic stenosis, whereas it is either narrow or reversed in aortic stenosis.

NON-EJECTION CLICKS

The term non-ejection click (NEC) is applied to systolic sounds occurring at the AV valves in prolapse of mitral or tricuspid valves due to myxomatous degeneration of the valve.

In a normal mitral valve and left ventricle, the mitral valve fits into the left ventricle like a cone. This conical shape of the mitral valve is a mechanical advantage as the force of ventricular contraction is mostly expended on the outflow rather than the inflow. With a flat curtain-like closure of the valve or prolapse, the valve faces the brunt of left ventricular pressure.

Mitral valve prolapse

Mitral valve prolapse is a common clinical syndrome due to diverse pathogenic mechanisms affecting the leaflets, chordae tendineae, papillary muscle, and mitral annulus. It is known by various names:

- Mitral valve prolapse syndrome
- Systolic click murmur syndrome
- Barlow syndrome
- Billowing mitral cusp syndrome
- Redundant cusp syndrome
- Floppy valve syndrome
- Myxomatous mitral valve

It affects 3–5 per cent of the population. Overdiagnosis by echocardiography is particularly common.

Determinants of first heart sound–non-ejection click interval

- Left ventricular end diastolic volume
- Rate of left ventricular ejection

The timing of the non-ejection click coincides with the maximal prolapse of the mitral valve into the left atrium. The click may be single or multiple, with or without mitral regurgitation.

Based on the degree of abnormality of the valve, the prolapse occurs at a

particular end diastolic volume of the ventricle. This is called the click volume. The click volume for individual patients is constant unless the lesion progresses. The ventricular end diastolic volume and the rate of ejection are the important determinants of S1–non-ejection click (S1–NEC) interval. All the maneuvers that decrease the ventricular end diastolic volume increase the prolapse and the click is earlier and louder. On the other hand, the maneuvers that increase the volume of the ventricle reduce the degree of prolapse and the click occurs later and reduces in intensity or disappears.

The clicks can be single or multiple, persistent or intermittent. They are best heard at the apex and being of high frequency are better heard with the diaphragm of the stethoscope. If loud they can be heard in other areas. The murmur is late systolic and characteristically changes in length with posture. It is usually best heard at the apex but sometimes better heard at the lower left sternal border (Fig. 21.10). In contrast to the murmur of rheumatic mitral regurgitation, the murmur of mitral valve prolapse is relatively rough and rasping in character. Mitral valve prolapse (MVP) is often mistaken for a variety of other conditions. In view of chest pain and electrocardiographic ST–T changes, MVP is often mistaken for coronary artery disease. This mistake is compounded by the exercise test, which is commonly false positive in MVP. While MVP generally carries a good prognosis, the presence of coronary artery disease has serious implications. A patient with MVP may present with a wide variety of features and one should look for MVP in all these clinical situations.

All the maneuvers which decrease in left ventricular size results in movement of the click towards S1 and which increase in size vice versa. Normally the chordae prevent the leaflets from prolapsing into the atrium because of tensing movement

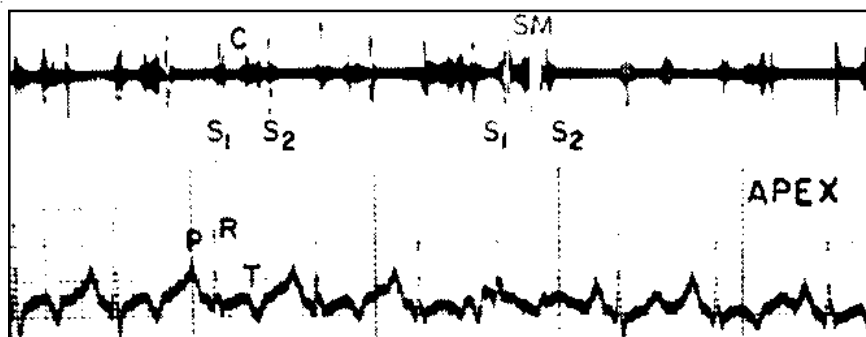


Fig. 21.10: Intermittent click murmur in mitral valve prolapse

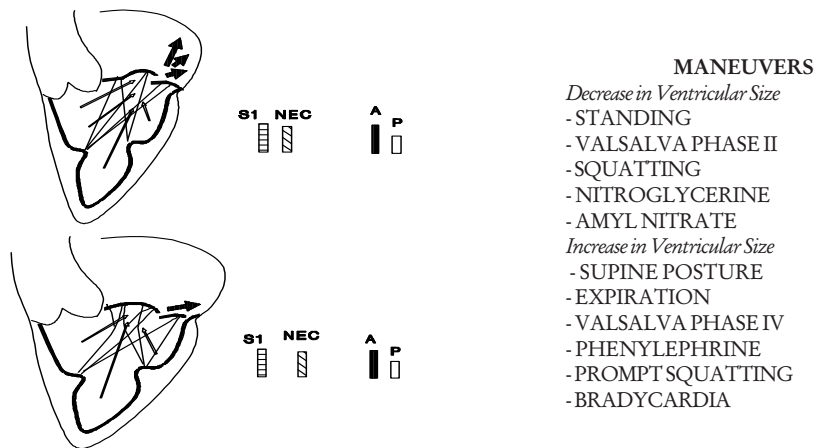


Fig. 21.11: Effect of various maneuvers on S1-NEC interval

due to (contraction of papillary muscles). When the chordae are long and redundant as in a myxomatous valve, or the papillary muscle function is impaired, the leaflets lose their support and prolapse into the atrium. As the chordae are stretched with increase in ventricular size, the amount of prolapse decreases. The opposite mechanism operates when the ventricle becomes small.

Situations in which mitral valve prolapse could occur are:

- Young patient with chest pain
- Unexplained ST-T alterations in inferolateral leads with or without chest pain
- Recurrent palpitations or unexplained atrial or ventricular arrhythmias

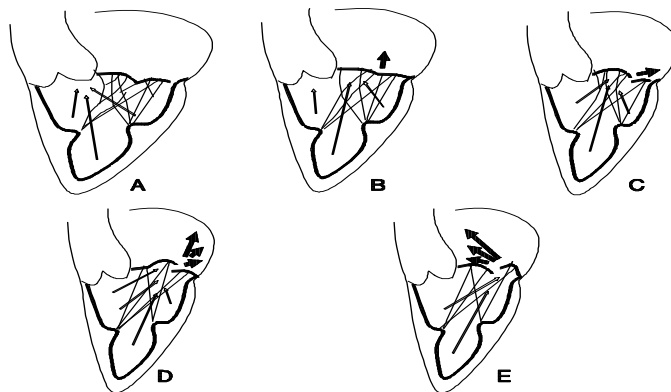


Fig. 21.12: Relationship of ventricular size to the degree of valve prolapse

EJECTION AND NON-EJECTION CLICKS

Table 21.5: Alterations in S1-NEC interval: effect of maneuvers

<i>Maneuvers</i>	<i>S1-NEC</i>	<i>S1-M</i>
<i>Decrease in ventricular size</i>		
Standing		
Valsalva phase II		
Squatting	Decreased	Decreased
Nitroglycerine		
Amyl nitrate		
<i>Increase in ventricular size</i>		
Supine		
Expiration		
Valsalva phase IV	Increased	Increased
Phenylephrine		
Prompt squatting		

(Phenylephrine increases the S1-NEC and S1-M interval and also increases the intensity of the murmur. S1-NEC = First heart sound – non-ejection interval; S1-M = First heart sound murmur interval.)

- Any patient with syncope
- Any patient with fever
- Young patient with stroke
- All patients with mitral regurgitation
- All patients with atrial septal defect (Fig. 21.14)
- All patients with Marfan syndrome
- All patients with chest deformity

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In all the above situations, auscultation should be performed as a matter of routine, with the patient in standing position.

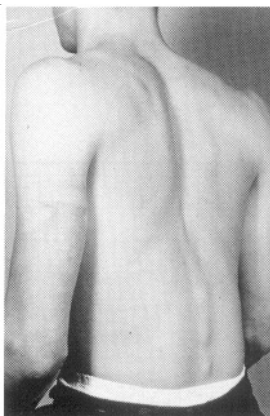


Fig. 21.13: Straight back

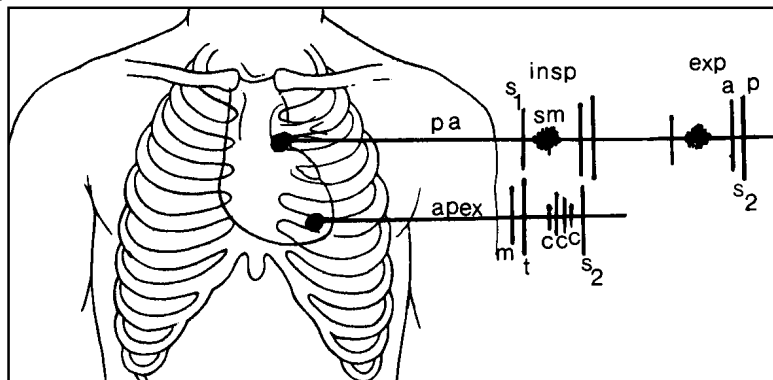


Fig. 21.14: Atrial septal defect with mitral valve prolapse

APPROACH TO PATIENT WITH CLICK SOUND IN SYSTOLE

When an extra sound is heard in systole, one should answer the following questions (see also Table 21.6).

- Is it a systolic click or some other sound mistaken for it?
- If it is a systolic click, is it an ejection click or a non-ejection click?
- If non-ejection click, is there associated mitral regurgitation?
- If ejection click, is it aortic or pulmonary?
- If aortic, is it vascular or valvular?
- If pulmonary, is it vascular or valvular?
- If pulmonary valvular click, is it due to mild, moderate or severe pulmonic stenosis?

The loud first sound and split first sound may be mistaken for an ejection click. The first sound is best audible at the apex and the split first sound is heard only at the tricuspid area.

Table 21.6: Approach to a patient with clicking sound in systole

<i>Finding</i>	<i>Inference</i>
Changes significantly with posture	Non-ejection click
If NEC	Look for the murmur of MR
No change with posture	Ejection click either aortic or pulmonary
Best heard at aortic area but is widely audible at LSB and apex	Aortic ejection click
If aortic, A2 is normal or accentuated, second heart sound normally split	Aortic vascular click
If aortic, A2 is diminished, S2 is reversibly split	Aortic valvular ejection click
Best heard at pulmonary area, not widely audible	Pulmonary vascular or valvular click
Loud P2, palpable pulmonary artery	Pulmonary vascular click
Wide split S2, diminished P2, impalpable pulmonary artery with systolic thrill at pulmonary area	Pulmonary valvular click, mild to moderate, or severe
Better heard during expiration in standing posture	Moderate or severe pulmonic stenosis

THE OPENING SNAP

The opening of the normally thin atrioventricular valves is noiseless. However, in disease the opening of these valves is associated with clicking noises called opening snaps. The mitral and tricuspid valves, when stenosed cause, opening snaps though there are many other causes of clicky or high-frequency noises in early diastole:

Mitral

- Mitral stenosis
- Mitral regurgitation (rare)
- Mitral valve prolapse

Tricuspid

- Tricuspid stenosis
- Ebstein's anomaly of tricuspid valve
- Functional tricuspid stenosis as in atrial septal defect

Tumoursounds

- Left atrial myxoma
- Right atrial myxoma
- Aneurysm of interventricular septum as in ventricular septal defect

Pericardial knock of constrictive pericarditis

Though the opening snap can be recorded in the conditions mentioned above, for all practical purposes, a clinically audible opening snap almost always means mitral stenosis. In mitral stenosis, the opening snap is due to thickening of the valve with a doming motion towards the left ventricle due to high pressure in the left atrium.

The various mechanisms are (Fig. 21.15):

- Thickening of the valve
- High pressure in the left atrium
- Doming of the stenotic valve (anterior leaflet)

The time at which the opening snap occurs is a function of the left atrial pressures. The higher the left atrial pressures, the earlier the opening snap. If the left atrial pressures are lower, the opening snap may occur later. The second heart sound–opening snap interval (S2–OS) is used to assess the severity of mitral stenosis. It is inversely related to the severity of mitral stenosis. The shortest S2–OS interval possible is 0.04 seconds and the longest is 0.12 seconds. At a heart

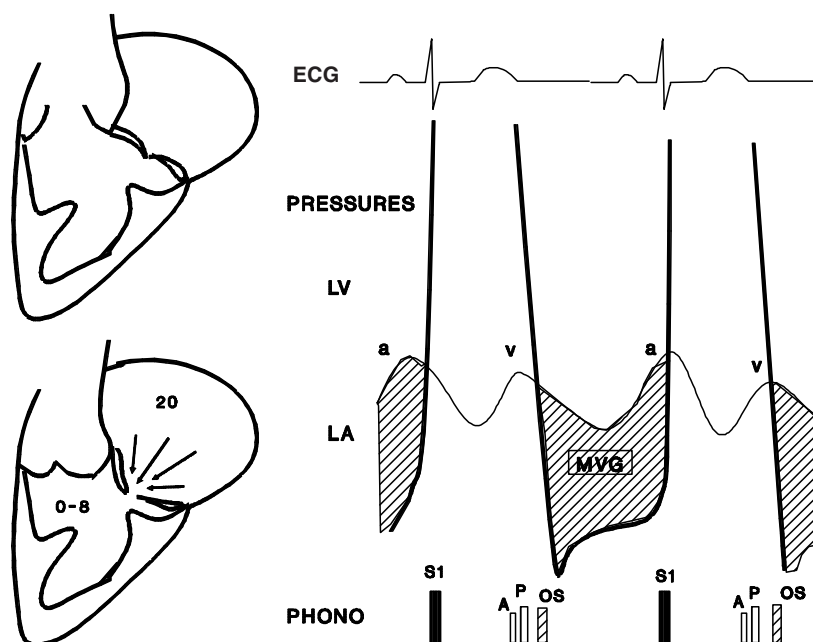


Fig. 21.15: Mechanism of mitral opening snap

rate of 70–90/min, a mild mitral stenosis has a S2–OS interval of 0.12 seconds, a moderate mitral stenosis 0.08 seconds and a severe mitral stenosis will have a S2–OS of 0.04 seconds.

In mitral stenosis, the leaflets are thickened and open with a doming motion. The left atrial pressures are high at the onset of diastole and result in an abrupt opening of the valve when the atrial pressure exceeds the ventricular pressure. This results in the classical opening snap. A calcified valve does not result in opening sound since the opening excursion is markedly restricted.

The left atrial (LA) pressure is higher with increasing severity of mitral stenosis. Hence, the crossover point of pressures, that is, the point where the LA pressure exceeds the LV pressure, occurs earlier and thus closer to the A2. Thus, the A2–OS interval has an inverse correlation with the severity of mitral stenosis in most situation (Fig. 21.16). The limitations of this correlation are discussed below.

Though the S2–OS is generally reliable in the estimating severity of mitral stenosis, it may not be reliable in situations where the diastole is shortened or prolonged with changes in heart rates, earlier or later occurrence of aortic valve closure, or elevation of LVEDP, or significant changes in cardiac output (Table 21.7).

EJECTION AND NON-EJECTION CLICKS

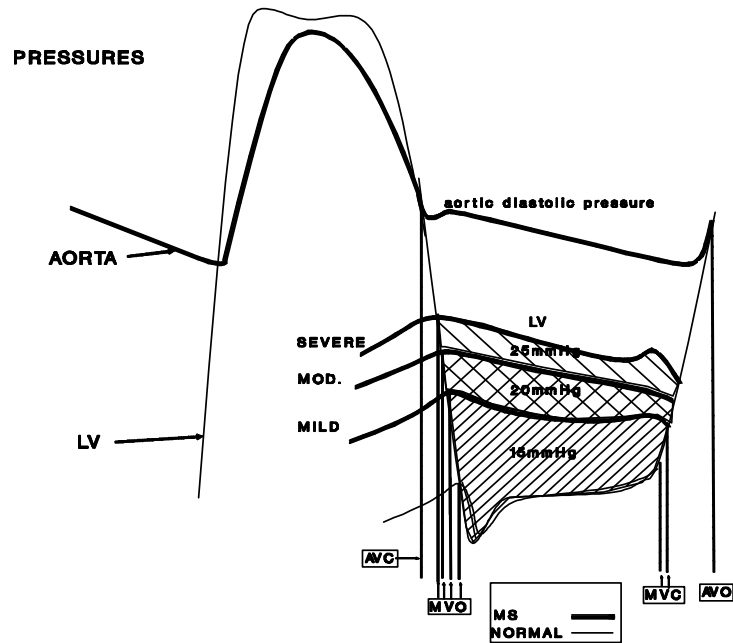


Fig. 21.16: Relationship of severity of mitral stenosis to S2-OS interval

Mechanisms altering the interval

- Alteration in heart rate
- Alteration in left atrial pressure
- Alteration in left ventricular end-diastolic pressure
- Alteration in aortic pressure
- Conditions affecting the velocity of mitral valve opening
- Aortic regurgitation
- Decreased left ventricular compliance
- Mitral valve calcification

Table 21.7: Second heart sound–opening snap interval in various degrees of mitral stenosis

Degree of mitral stenosis	S2-OS	LA pressure	MVA
Mild mitral stenosis	0.12 sec	15 mmHg	1.5–2.5 cm ²
Moderate mitral stenosis	0.08 sec	20 mmHg	1.0–1.5 cm ²
Severe mitral stenosis	0.04 sec	25 mmHg	< 1.0 cm ²

Table 21.8: Conditions where second heart sound–opening snap is unreliable

<i>Condition</i>	<i>Shortened/widened</i>	<i>Mechanism</i>
Tachycardia	Shortened	Abbreviation of diastole
Bradycardia	Prolonged	Prolonged diastole
Hypertension	Widened	Early aortic closure
Aortic stenosis	Shortened/widened	Delayed aortic closure
Aortic regurgitation	Widened/shortened	Early aortic closure
Low cardiac output	Widened	Lower left atrial pressure
Severe RVF		
Severe TR		
Severe PAH		
Increased LVEDP	Widened	Obliteration of transmitral gradient
Coronary artery disease		
Cardiomyopathy with systolic or diastolic dysfunction		

The opening snap is virtually pathognomonic of mitral stenosis. An audible opening snap distinguishes mitral stenosis from other conditions simulating it. If an opening snap is not heard in the setting of mitral stenosis, a calcified valve, mitral regurgitation, severe aortic regurgitation, aortic stenosis, coronary artery disease or any condition with left ventricular failure should be considered. In general, a narrow S2–OS interval always indicates tight mitral stenosis. However, when the interval is wide, tight mitral stenosis is not ruled out.

Missing opening snap in mitral stenosis

This could be due to:

- Severely calcified mitral valve
- Mitral regurgitation (significant)
- Aortic regurgitation (severe)
- Aortic stenosis (severe)
- Coronary artery disease with left ventricular dysfunction
- Any condition with associated left ventricular failure
- Very close S2–OS (< 30 msec)
- Auscultatory incompetence
- OS heard but mistaken for wide split second heart sound

With milder degree of calcification, the opening snap may still be heard. With all the other lesions, the lesion has to be severe enough to obliterate the opening snap. For the information of the junior student and the general practitioner the commonest cause for absence of opening snap is the inability to hear rather than a real absence. Even when audible, the second heart sound–opening snap is mistaken for a split second heart sound.

Clinical Recognition of OS and S2–OS Interval

The opening snap is a high-frequency sharp click best heard with the diaphragm of the stethoscope. It is heard internal to the apex, left sternal border and occasionally in the pulmonary area. As the murmur of the mitral stenosis is louder at the apex, the opening snap is difficult to detect at the apex. Exercise may bring it out when it is not heard at rest. It does not change significantly with respiration. When it is heard at the pulmonic area, it may be confused for a wide split second heart sound. Audibility over a wide area and the last of the two components being fainter differentiate the opening snap from the wide split second sound. The best way for a student to practice listening for the opening snap is take a patient with classic mitral stenosis with easily audible opening snap, and auscultate for 20–30 minutes until you get an audible feel of it. Then move away from the site of best audibility to the site of least audibility when it becomes fainter. This is the type of sound one hears in a ‘difficult’ patient with mitral stenosis (silent mitral stenosis).

The S2–OS interval is best appreciated by the knowing and appreciating the time intervals of other sounds. For example, the shortest S2–OS interval (0.04 seconds) sounds like a split second heart sound. The widest S2–OS sounds like an early third heart sound as in severe heart failure, severe mitral regurgitation or the pericardial knock of constrictive pericarditis (0.12 seconds). The moderate S2–OS interval sounds like the wide split second heart sound of atrial septal defect. The sounds following the second heart sound and their time intervals are:

S2 split (inspiration)	0.04–0.05 sec
S2 single (expiration)	< 0.03 sec
S2 wide split	0.06–0.12 sec
S2–OS interval	0.04–0.12 sec
S2–pericardial knock	0.10–0.12 sec
S2–S3 interval (pathological)	0.12–0.16 sec
S2–S3 normal (children)	0.12–0.20 sec

CONDITION	S1	A	P	INTERVAL (mSec)
Normal S2 split(Insp.)	■	▨	▨	40-50
Normal S2 split (Exp.)	■	▨	▨	<30
S2 wide split	■	▨	▨	60-120
Pathological S2-S3 interval	■	▨	▨	120-160
Physiological S2-S3 interval	■	▨	▨	120-200
S2-Pericardial knock	■	▨	▨	100-120
S2-OS interval	■	▨	▨	40-120

Fig. 21.17: Sounds around A2 mistaken for OS

Sounds occurring around A2 can be mistaken for an opening snap (Fig. 21.17). These include a third heart sound (S3), pericardial knock and P2. The normal intervals between A2 and these sounds are given in Fig. 21.16. We can observe from the values given, that there can be significant overlap between various intervals.

In mitral stenosis the opening snap can give the following information:

- Diagnosis of mitral stenosis
- Differential diagnosis of mitral stenosis from conditions simulating it
- Assessment of severity of mitral stenosis
- Detection of complications like calcifications
- Recognition of associated disorders

If the S2–OS interval is wide but the patient has significant symptoms, one should consider the possibility of left atrial myxoma as a cause of mitral valve obstruction. In contrast to the audible opening snap of mitral stenosis or TS, ‘phonographically recordable’ opening snaps are described in a variety of conditions (Table 21.9).

EJECTION AND NON-EJECTION CLICKS

Table 21.9: 'Recordable' opening snaps in the absence of AV valve stenosis

<i>Mitral opening snap</i>	<i>Tricuspid opening snap</i>
Mitral regurgitation (may be audible)	Atrial septal defect
Mitral valve prolapse (may be audible)	Tricuspid regurgitation
Ventricular septal defect	
Patent ductus arteriosus	
Heart block (second and third degree)	
Tricuspid atresia	
Tetralogy after shunt operation	
Thyrotoxicosis	
Hypertrophic obstructive cardiomyopathy	
Mitral valve prolapse	

The basic mechanism in most of these conditions is increased flow across the AV valve. The degree of thickening and redundancy may play a role in mitral valve prolapse. Valve thickening is also described as the mechanism in hypertrophic cardiomyopathy.

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DIFFERENTIAL DIAGNOSIS OF SOUNDS

In nature, there can never be two things which are exactly alike.

Leibnitz

The various sounds in systole and diastole can be represented as shown in Fig. 21.18.

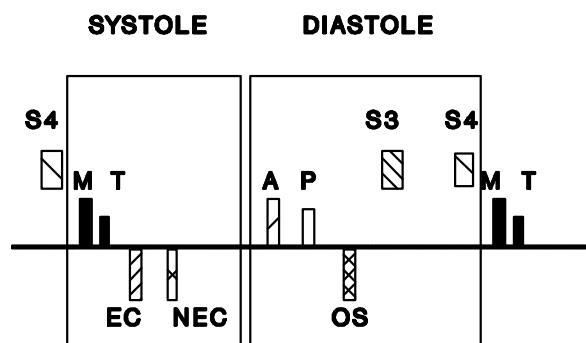


Fig. 21.18: Clustering of additional sounds around S1 and S2

Table 21.10: Recognition of sounds

<i>Confusion around first heart sound (S1)</i>	<i>Confusion around second heart sound (S2)</i>
Split S1 S1–ejection click S1–non-ejection click S4–S1	S2 split S2–OS S2–S3 Non-ejection click–S2 (rare)

For the purpose of clarity (confusion!), this can be divided into two groups:

- Confusion around S1
- Confusion around S2

The general principles by which one sound is differentiated from the other are (Table 21.10):

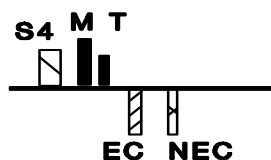
- Site of best audibility of a sound
- Localized or widely audible
- Character of the sound
- Palpability
- Relation to a physiological act
- Accompanying features

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Right sided ejection clicks are generally not confused with other sounds as they are localized to the pulmonary area. These principles can be applied to each of the sounds (Tables 21.11, 21.12).

CONFUSION AROUND

S1



S2

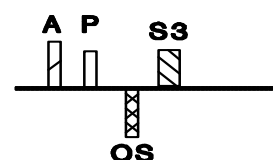


Fig. 21.19: Differentiating confusion around S1 and S2

EJECTION AND NON-EJECTION CLICKS

Table 21.11: Sounds around first heart sound (confusion around S1)

<i>Feature</i>	<i>Split S1</i>	<i>S1-EC</i>	<i>S1-NEC</i>	<i>S4-S1</i>
Site of best audibility	Tricuspid area	Base	Apex	Apex
Localized/ widely audible	Localized	Widely audible	Widely audible	Localized
Character	Low frequency	High frequency, sharp, clicky	High frequency, sharp, clicky	Low frequency
Palpability	No	No	No	Often palpable
Relation to physiological act	Best audible during inspiration	No change in Aortic click	NEC loud and earlier with standing	Isometric hand grip increases S4
Associated features	Normal RBBB ASD Ebstein's anomaly	Aortic stenosis Bicuspid aortic valve	Mitral valve prolapse	CAD Cardiomyopathy LVH with HTN

Table 21.12: Sounds around second heart sound (confusion around S2)

<i>Feature</i>	<i>S2 split</i>	<i>S2-OS</i>	<i>S2-S3</i>	<i>NEC-S2</i>
Site of best audibility	Pulmonary area	Internal to apex	Apex	Apex
Localized widely audible	Localized	Widely audible	Localized	Variable
Character	High pitch	High pitch	Low pitch	High pitch
Palpability	P2 may be palpable	Not palpable	Palpable	Not palpable
Relation to physiological act	Inspiration increases	Respiration has no effect	Expiration increases	Standing posture increases
Associated features:	Normal RBBB ASD Ebstein's anomaly	Mitral stenosis	Normal Heart failure MR	Mitral valve prolapse

Though it is unusual for the non-ejection click to be delayed enough to be close to the second heart sound, errors are common when this happens.

PRACTICE IMPLICATIONS

A child with valvular aortic stenosis should always have an ejection click. Absence of it makes the diagnosis of aortic stenosis suspect, or the stenosis is at another site, for example, subvalvular or supra-valvular. This physical sign may sometimes be more reliable than some echocardiograms. The following case summary illustrates this.

Case summary

A 9-year-old girl was referred for evaluation of two echocardiogram reports done within the same week. One of the reports read congenital bicuspid aortic valve, peak systolic gradient of 104 mmHg, left ventricular hypertrophy. Another opinion was sought with a repeat echocardiogram/Doppler. This time the report read congenital bicuspid aortic valve, superior and inferior orientation, peak systolic gradient 57 mmHg, mild concentric left ventricular hypertrophy. The cause for referral was the controversy over the gradients across the aortic valve which was bicuspid.

Five years earlier, she was detected to have patent ductus arteriosus with left to right shunt and congenital bicuspid aortic valve after two echocardiograms. Cardiac catheterization confirmed the diagnosis of patent ductus arteriosus and aortic stenosis. The peak systolic gradient across the aortic valve was 10 mmHg. Surgical closure of the ductus was done. The child was followed up every year by echocardiogram and Doppler.

The child was asymptomatic and the physical examination revealed features of aortic stenosis. The most remarkable feature was absence of an ejection click thus making Valvular aortic stenosis unlikely. Yet another echocardiogram was ordered with a request to look for subvalvular membrane as a cause for aortic stenosis. This time the echocardiographer could see a thick subvalvular membrane close to the aortic valve. The aortic valve was normal and tricuspid.

This summary illustrates the value of clinical examination in directing laboratory tests and also the importance of the absence of ejection click in a child with aortic stenosis.

"You've heard about me, Mr. Holmes," she cried, "else how could you know all that?"

"Never mind." said Holmes, laughing. "It is my business to know things. Perhaps I have trained myself to see what others overlook. If not, why should you come to consult me?"

22 Heart Murmurs

Murmurs, unlike sounds, are prolonged vibrations. They are due to disturbance in blood flow, that manifests as turbulence. Turbulence is defined as an irregular state of motion in which velocity and pressure show a random variation in relation to space. The site of maximum intensity of a murmur generally corresponds to the site of the turbulence (for example, the root of the great vessels in aortic stenosis and pulmonic stenosis, the left atrium in mitral regurgitation and the cavity of the left ventricle in aortic regurgitation).

Timing of murmurs

Murmurs are classified by their timing as systolic, diastolic and continuous. Figs. 22.1 and 22.2 show the various types of murmurs and Table 22.1 shows the classification.

Table 22.1: Classification of murmurs

Category	Definition
<i>Systolic murmurs</i>	<i>Starts with or after S1, and ends before or at S2</i>
Ejection systolic	Starts after S1, ends before S2 of that side (A2 or P2)
Pansystolic	Starts with S1 and ends with S2 of that side (A2 or P2)
Late systolic	Starts after S1, ends with S2 of that side (A2 or P2)
Early systolic	Starts with S1, does not reach S2
<i>Diastolic murmurs</i>	<i>Starts with or after S2, ends at or before S1</i>
Early diastolic	Starts with S2 (A2 or P2), duration in diastole is variable
Mid-diastolic	Starts after S2, ends before S1
Late diastolic	Starts late after S2 and extends to the S1 of that side (mitral, tricuspid)
Holodiastolic	Early diastolic murmurs occupying whole of diastole from S2 to S1
Continuous murmur (CM)	Beginning anywhere in systole, continues into diastole, encompassing S2

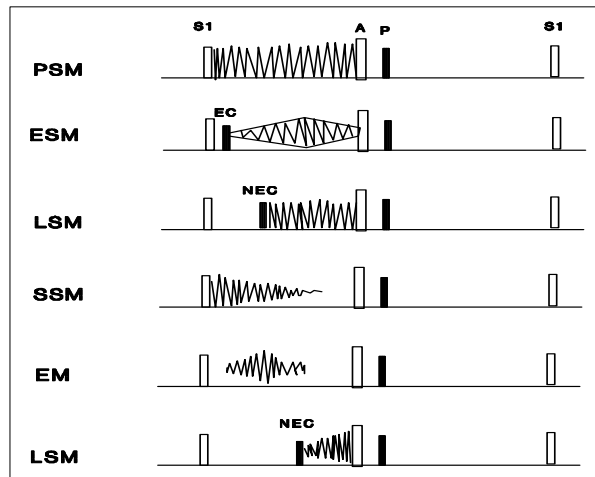


Fig. 22.1: Definitions of systolic murmurs

PSM: Pansystolic murmur, starts with first heart sound and ends with second heart sound of that side (A2 or P2); ESM: Ejection systolic murmur, starts after first heart sound and ends with or before second heart sound of that side; LSM: Late systolic murmur, starts well after first heart sound and ends with second heart sound of that side; SSM: Short systolic murmur, may start with or after first heart sound and ends well before second heart sound; EM: Ejection murmur, starts after first heart sound and ends well before second heart sound.

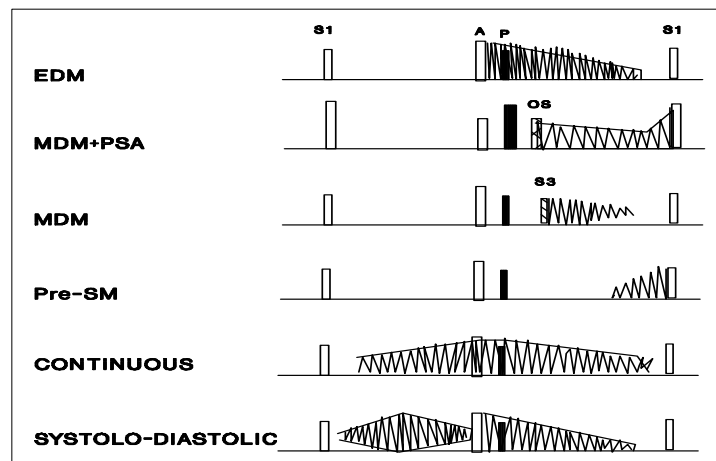


Fig. 22.2: Definitions of diastolic and continuous murmurs

EDM: Early diastolic murmur, starts with second heart sound of that side and ends at a variable interval; MDM: Mid-diastolic murmur, starts after second heart sound and ends with or before first heart sound; Pre-SM: Pre-systolic murmur, occurs just before systole ending in first heart sound and is usually due to atrial booster; Continuous murmur: Begins anywhere in systole and continues into diastole encompassing second heart sound; Systolo-diastolic: Combination of ejection or pansystolic murmur with early diastolic murmur and can be mistaken for a continuous murmur.

SYSTOLIC MURMURS

Systolic murmurs are principally classified as ejection systolic and regurgitant murmurs.

Grading of murmurs

Systolic murmurs are graded, based on their intensity and the presence or absence of thrill. As systolic murmurs often occur even in the absence of organic heart disease, it is important to differentiate organic murmurs from functional murmurs. Murmurs above grade 3 are usually organic; grade 4 murmurs are almost always organic. As the presence of a thrill makes significant implications in the interpretation of murmurs, one should be careful in eliciting this physical sign. The palpable vibrations of the pulmonic sound in the pulmonary area and the first heart sound at the apex are often mistaken for a thrill. A useful clue is that in the presence of a thrill, the accompanying murmur is always loud. If the murmur is faint, an associated thrill is highly unlikely. The original grading was applied for systolic murmurs whose origin can be organic or functional. Diastolic murmurs are not generally graded because the very presence of a diastolic murmur signifies heart disease. Some clinicians grade diastolic murmurs by their length. The current preference is to grade both systolic and diastolic murmurs by the same method.

Functional murmurs may be grade 4 in certain situations. The causes could be:

- Venous hum
- The ejection systolic murmur in the carotid in severe aortic regurgitation
- Mitral mid-diastolic murmur in severe mitral regurgitation
- Early diastolic murmur of pulmonary hypertension (especially in primary PAH)
- Ejection systolic murmur at the pulmonary area in atrial septal defect

Table 22.2: Grading of systolic murmurs

<i>Grade of murmur</i>	<i>Basis</i>
Grade 1	Faint murmur heard on careful auscultation
Grade 2	Easily audible but not loud
Grade 3	Easily audible, loud murmur but no thrill
Grade 4	Murmur with a thrill and is usually loud
Grade 5	Murmur audible with the stethoscope partially applied to the chest
Grade 6	Murmur audible with the stethoscope half an inch to an inch away from the chest

The diastolic thrill in pulmonary regurgitation with pulmonary arterial hypertension is often unexpected and the murmur is mistakenly timed as systolic.

Classification

Ejection systolic murmurs are of two kinds: organic, and functional or innocent.

Organic systolic murmur

LV outflow obstruction

- Valvular
- Subvalvular
 - Fixed
 - Dynamic (HOCM)
- Supravalvular

RV outflow obstruction

- Valvular
- Subvalvular
 - Infundibular
 - Double chambered RV
- Supravalvular

Functional or innocent systolic murmur

LV outflow

- Increased flow
 - Aortic regurgitation
 - Hyperkinetic states
 - Complete heart block
- Dilation of ascending aorta
 - Aneurysm
 - Aortitis
 - Systemic hypertension

RV outflow

- Increased flow
 - Left to right shunts (ASD, VSD)
 - Hyperkinetic states

HEART MURMURS

- Idiopathic dilatation
- Pulmonary artery hypertension
- Innocent systolic murmurs in children
- Chest wall and mediastinal factors
 - Chest deformities
 - Thin chest, straight back syndrome, pectus excavatum
 - Kyphoscoliosis, pulmonary fibrosis

The term organic is used to refer to a structural defect responsible for the murmur. The terms functional and innocent are used to indicate a non-organic cause for the murmur. A functional murmur by definition should subserve a function like increased flow across the aortic valve as in the ejection systolic murmur of severe aortic regurgitation. For this reason the terms innocent and functional murmurs should not be used interchangeably. The term 'innocent' is used to refer to murmurs that normally occur in the absence of anatomic or physiologic abnormalities of the heart or circulation. They can occur at any age but are particularly more common in children. Innocent murmurs are more common on the right side of the heart across the RV outflow than on the left side. It is a good practice to routinely look for intrinsic heart disease if a murmur is heard across the left ventricular outflow in a young person.

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Innocent or normal murmurs are of two types: systolic and continuous:

Systolic

- Vibratory murmur (Still's murmur)
- Pulmonic systolic murmur
- Peripheral pulmonary systolic murmur
- Supraclavicular murmur
- Aortic systolic murmur
- Systolic mammary souffle

Continuous

- Venous hum
- Mammary souffle

EVALUATION OF A MURMUR

A murmur is the most important physical sign in the majority of valvular and congenital heart diseases. A systematic evaluation is essential to derive maximum information from this physical sign. Carefully analyzed, each component in the description of a murmur is valuable in drawing conclusions. The important features are:

- Site of best audibility
- Timing, configuration or shape
- Grading
- Length
- Character
- Selective conduction
- Relation to a physiological act or maneuver
- Accompanying features

Site

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Murmurs of right sided origin are generally not heard at the apex. However, when the right ventricle forms the apex, any of the right sided murmurs can be heard at apex. The ESM of calcific aortic stenosis in elderly patients with severe emphysema can be heard only at the apex due to two mechanisms:

- a) Severe emphysema does not allow any portion of the left ventricle to come into contact with chest wall except the region of apex.

<i>Murmurs heard at mitral area or apex</i>	
Murmurs heard only at apex	MDM of mitral valve origin Mild or trivial MR ESM of calcific AS in elderly with emphysema EDM of AR (rare) TR of Ebstein's anomaly of TGA
Murmurs best heard elsewhere but also heard at apex	EDM of AR ESM of AS MDM of tricuspid origin in ASD or TS Pansystolic murmur of TR Pansystolic murmur of VSD
Murmurs which are usually not heard at apex	ESM of PS EDM of pulmonary incompetence
Murmurs which are never heard at apex	None

HEART MURMURS

<i>Murmurs heard at tricuspid area (TA)</i>	
Murmurs heard only at TA	Tricuspid stenosis Mild tricuspid regurgitation Small ventricular septal defect
Murmurs best heard elsewhere but also heard at TA	Mitral regurgitation Aortic regurgitation Aortic stenosis Pulmonic stenosis Ventricular septal defect Mitral stenosis (rare)
Murmurs which are generally not heard in TA	Mitral stenosis
Murmurs which are never heard at TA	None

<i>Murmurs heard at left sternal border (LSB) (third space)</i>	
Murmurs best heard at the LSB	Pansystolic murmur of VSD ESM of AS EDM of AR ESM of infundibular PS
Murmurs best heard elsewhere but also along LSB	ESM of valvular PS TR MR
Murmurs generally not heard along LSB	MS
Murmurs never heard along LSB	None

<i>Murmurs heard at pulmonary area (left second space)</i>	
Murmurs best heard at the pulmonary area	ESM of pulmonic stenosis Flow murmur of pulmonary origin in ASD Continuous murmur of PDA VSD Pulmonary incompetence
Murmurs best heard elsewhere but also heard at pulmonary area	AS VSD MR AR
Murmurs which are present but unusual at pulmonary area	MS TS
Murmurs which are never heard at pulmonary area	None

<i>Murmurs heard at aortic area (right second space)</i>	
Murmurs best heard at the aortic area	Valvular AS Aortic valve sclerosis Subvalvular membrane close to the aortic valve AR of aortic root origin
Murmurs best heard elsewhere but also heard at aortic area	All levels of LV outflow obstruction MR of mitral valve prolapse AR of any etiology VSD
Murmurs rarely heard at the aortic area	PS TR
Murmurs never heard at the aortic area	MS (almost never)

- b) The loss of jet effect in a severely calcific valve, which prevents conduction of the murmur to the carotids.

The mitral diastolic murmur is occasionally heard at the tricuspid area and along the left sternal border.

The commonest murmur heard along the left sternal border is the pansystolic murmur of ventricular septal defect.

It is extremely unusual for the murmur of mitral stenosis to be audible at the pulmonary area but is possible in a rare case.

Timing

Timing of the murmur gives clues as to the site of origin of the murmur.

Ejection systolic murmurs: The ejection systolic murmur by definition means a murmur starting some time after the first heart sound and reaching peak by mid-systole or later and ending before the second heart sound. The moment the ventricle begins its contraction, first heart sound occurs due to closure of the AV valves but takes a while (isovolumic contraction time) to exceed the pressure in the aorta to open the aortic valve and start ejection (Table 22.3).

Ejection systolic murmur-like murmurs may occur at other sites in some special situations as listed above. However, the classic mid-systolic peaking and late systolic tapering is rare.

HEART MURMURS

Table 22.3: Localizing value of ejection systolic murmurs

<i>Site</i>	<i>Mechanism</i>
LV outflow	Obstruction
RV outflow	Increased flow Increased pressure Dilatation of vessel beyond
<i>ESM-like murmurs</i>	
Small muscular VSD	Valve-like mechanism
Large VSD	Equalization of pressures/murmur may actually be across the RV outflow
Acute MR/TR (early systolic)	Small atria with obliteration of pressure in later systole
MR/TR (short systolic)	Milder lesions
<i>Patent ductus arteriosus</i>	
PAH	Equalization of pressures
Large ductus	Equalization of pressures
Long narrow ductus	Valve-like mechanism

Pansystolic murmurs: A pansystolic murmur is a reflection of a pansystolic pressure difference between the two chambers in the heart. Only three sites in the heart are capable of pansystolic murmurs, the ventricular septal defect, mitral regurgitation and tricuspid regurgitation. In actual practice, the murmur of the ductus may be mistaken for that of a ventricular septal defect when the diastolic component of the continuous murmur is absent, as in a very large ductus with either hyperkinetic or fixed pulmonary arterial hypertension. In severe aortic stenosis or pulmonic stenosis, the very long ejection systolic murmurs may easily be mistaken for a pansystolic murmur of either ventricular septal defect or mitral regurgitation. The accompanying features of outflow obstruction help in the differential diagnosis.

Late systolic murmurs: Late systolic murmurs arise at the mitral valve due to mitral valve prolapse with mitral regurgitation.

Diastolic murmurs: These are of two kinds:

a) Mid-diastolic/pre-systolic murmurs: These murmurs are called mid-diastolic because they occur some time after the closure of semilunar valve and before the opening of the AV valve (isovolumic relaxation time). The pre-systolic murmurs are due to

Table 22.4: Localizing value of pansystolic murmurs

<i>Site</i>	<i>Mechanism</i>
VSD	Persistent high pressure difference between LV and RV
MR	Persistent high pressure difference between LV and LA
TR	Persistent high pressure difference between RV and RA as in severe PAH or PS
<i>Pansystolic-like murmurs</i> PDA (large)	High PA pressures obliterate the diastolic component of CM
Long ESMs of severe AS or PS	Very high gradients, with prolonged gradient and murmur

Table 22.5: Localizing value of mid-diastolic/presystolic murmurs

<i>Site</i>	<i>Mechanism</i>
Mitral/tricuspid valve	Forward flow of blood occurs at these valves only after the closure of aortic and pulmonary valves (A2, P2) and opening of mitral and tricuspid valves. The time interval is called isovolumic relaxation time.
<i>Mid-diastolic like murmur</i> Pulmonary regurgitation with normal PA pressure at the pulmonary area	With low pressure pulmonary regurgitation produces turbulence later in the diastole across the RV outflow

the atrial contraction maintaining flow in pre-systole or the last part of diastole. Only two sites in the cardiovascular system are capable of these murmurs, the mitral and tricuspid valves (Table 22.5).

The 'mid-diastolic-like early diastolic murmur' is heard in organic pulmonary regurgitation with normal pulmonary artery pressures.

b) Early diastolic murmurs (EDM): The early diastolic murmurs begin flush with semilunar valve closure of that side (aortic sound or pulmonic sound). They are related to backward flow of blood across the semilunar valves under high pressure. Only two sites in the circulation are capable of murmurs of this timing, aortic and pulmonary regurgitations (Table 22.6).

HEART MURMURS

Table 22.6: Localizing value of early diastolic murmurs

<i>Site</i>	<i>Mechanism</i>
Aortic valve regurgitation	Backflow of blood occurs flush with aortic component of second heart sound
Pulmonary regurgitation of PAH (Graham-Steell murmur)	Backflow of blood occurs flush with pulmonary component of second heart sound
<i>Early diastolic like mid-diastolic murmurs</i> Tricuspid flow murmurs	Tricuspid mid-diastolic murmurs are nearer to P2 than their counterparts on the left side

Table 22.7: Localizing value of a continuous murmur over the precordium

<i>Site</i>	<i>Mechanism</i>
<i>Aorta or systemic artery with another extracardiac site</i> PDA/AP window	Aorta to pulmonary artery with a systolic and diastolic pressure difference
Systemic arteriovenous fistula	Artery to vein communication with a large pressure difference continuously
<i>Aorta to one of the cardiac chambers</i> Aorta to RA Aorta to RV	RSOV to RA RSOV to RV

The tricuspid diastolic murmurs appear closer to the pulmonic sound, as the pulmonic sound occurs later than the aortic sound. Additionally, when the second heart sound is wide split as in atrial septal defect, the 'mid-diastolic murmur' appears even earlier.

Continuous murmurs: Continuous murmurs by definition start in systole and continue into diastole overlapping the second heart sound. The continuous murmur requires a communication between two sites in circulation, which have a continuous high pressure difference. This type of pressure relationship is possible only with a communication between two extracardiac sites or an extracardiac site with a cardiac chamber.

Length

The length of a murmur is generally a reflection of the duration of pressure difference between two sites in the cardiovascular system if a functional cause for

Table 22.8: Importance of length of the murmur

<i>Condition</i>	<i>Significance and mechanisms</i>
All stenotic lesions	Directly proportional to length
<i>Exceptions</i>	
Conditions which increase flow	Anemia, thyrotoxicosis, pregnancy, anxiety
Associated regurgitation	Adds to the amount of blood that has to be moved across the stenotic valve
Conditions which decrease flow	Heart failure, low cardiac output as in elderly people even without obvious heart failure, diuretics
Proximal severe lesion	Stenosis/regurgitation/shunt
Regurgitant lesions	Generally unreliable
Pansystolic murmurs (MR/VSD/TR)	Long murmur may be associated with lesion of any degree of severity
	Short murmur may be associated with any degree of severity as in acute MR
Early diastolic murmurs	
Aortic regurgitation	Length of the murmur is directly proportional to severity usually
	Exceptions
	Acute AR
	LVF
	Systemic hypertension
Pulmonary regurgitation	Due to PAH is longer and of high frequency
	With no PAH is shorter and of low frequency
Continuous murmurs	Short or long murmur may be associated with any degree of severity
	In arterial narrowing, the longer the murmur and higher the frequency, the greater the narrowing

a murmur is excluded. This is true in all stenotic lesions as in mitral stenosis, aortic stenosis, pulmonic stenosis or tricuspid stenosis. In lesions producing pansystolic murmurs, as in mitral regurgitation, ventricular septal defect, and tricuspid regurgitation, this rule is not applicable. This is because the very presence of these lesions results in pansystolic murmurs irrespective of the severity of the lesion. On the other hand, absence of expected pansystolic murmur has important implications in the evaluation of these lesions and will be discussed in detail under those conditions. The length of the early diastolic murmur of aortic regurgitation

generally correlates with the severity of aortic regurgitation, but relationship to length is not as direct as in stenotic lesions. The length of the murmur has no consistent correlation to the severity of lesions in regurgitant lesions. A short systolic murmur of mitral regurgitation or tricuspid regurgitation may indicate anything from a very mild to a very severe lesion or acute lesion. The accompanying features are important. In aortic regurgitation, the length of the murmur correlates better than in mitral regurgitation, but is not as reliable as in stenotic lesions. A short early diastolic murmur may be due to mild, moderate or severe aortic regurgitation with heart failure or acute severe aortic regurgitation.

Character

The character of the murmur is generally a clue to the lesion responsible. In general, murmurs with a high pressure difference between the two chambers are of high frequency or pitch, and those with a low pressure difference are of low frequency or pitch.

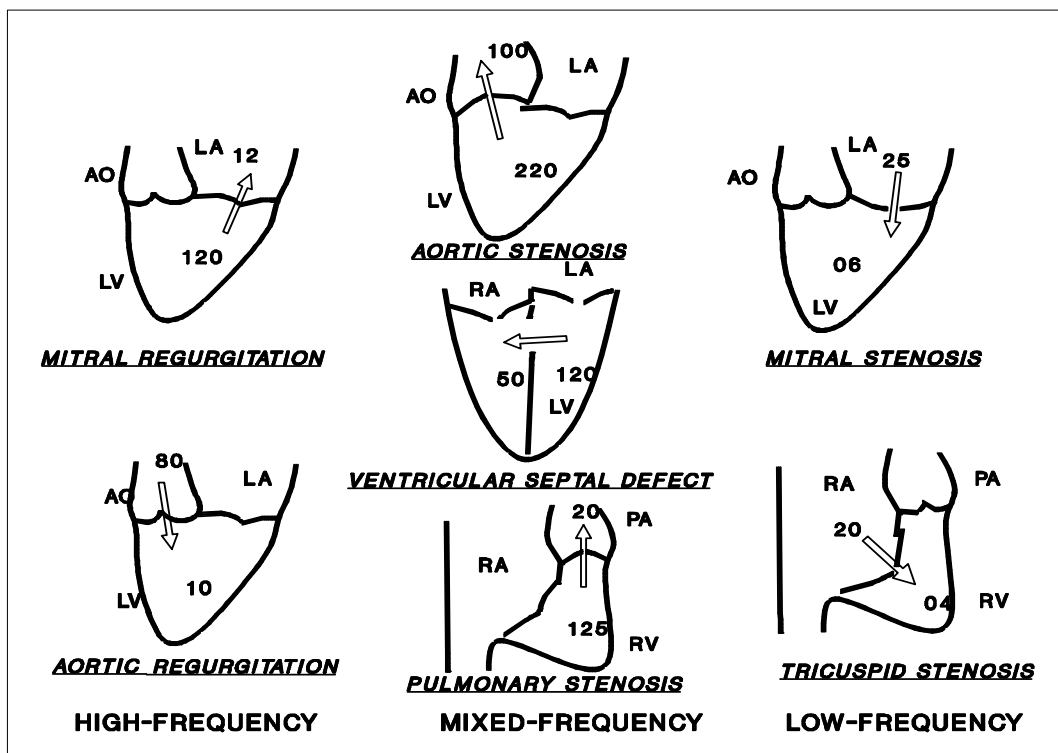


Fig. 22:3: Relation between pressure gradients and the frequency of the murmur generated

Table 22.9: Character of the murmur

<i>Feature</i>	<i>Mechanism</i>
<i>High frequency (high pitch)</i>	High pressure difference between two sites
Soft	MR (LV 120 to LA 12 mmHg)
Blowing	AR (Aorta 80 to LV 12 mmHg)
Musical	
<i>Low frequency (low pitch)</i>	Low pressure difference between two sites
Rough	MS (LA 20 to LV 5 mmHg)
Rumbling	TS (RA 15 to RV 5 mmHg)
<i>Mixed frequency</i>	High pressure difference between two sites
Rough	AS (LV 200 to Aorta 120 mmHg)
Harsh	PS (RV 120 to PA 25 mmHg)
	VSD (LV 120 to RV 25 mmHg)
	AR, PR (rare)

As a general rule, all regurgitant murmurs (mitral regurgitation, aortic regurgitation, tricuspid regurgitation, pulmonary regurgitation) are of high frequency and all stenotic murmurs are rough. The murmurs of AV valve stenosis (mitral stenosis and tricuspid stenosis) are of low frequency but the murmurs of semilunar valve stenosis are mixed in frequency. This combination of frequencies is a feature of aortic stenosis, pulmonic stenosis and ventricular septal defect. The low frequency or rough component of the murmur is best heard at the site of best audibility of the murmur. The high frequency or soft component of the murmur is more widely audible and is conducted to the other sites. This is the reason why the murmur of aortic stenosis is soft at the apex, and is mistaken for mitral regurgitation.

The character of the murmur depends on the pressure gradient across the defect, the pressure in the distal chamber and the nature of the defect. The murmur of aortic regurgitation may be rough in rare cases, be associated with a thrill and is usually due to a retroverted aortic cusp in aortic root disease as in syphilis or Marfan syndrome. The Graham Steell murmur of pulmonary arterial hypertension can occasionally be rough and may have a thrill as in some patients with primary pulmonary arterial hypertension or severe pulmonary arterial hypertension in association with patent ductus arteriosus. When these diastolic murmurs sound rough and have thrill, they can be mistaken for systolic murmurs.

HEART MURMURS

Table 22.10: Effect of physiological and pharmacological maneuvers on murmurs

<i>Murmur</i>	<i>Maneuver</i>
Valvular AS vs MR	MR murmur does not vary with cycle length, but AS varies Amylnitrite decreases MR Phenylephrine increases MR
Valvular AS vs HOCM	Valsalva maneuver, prompt squatting increase the murmur of HOCM
MR vs TR	Inspiratory increase of TR
MS vs Austin Flint murmur (A-F)	Amylnitrite increases MS but decreases or abolishes A-F Phenylephrine increases A-F
MS vs TS	Inspiration increases TS Expiration increases MS
Pure PS vs tetralogy of Fallot	Amyl nitrite decreases Tetralogy murmur
Rheumatic MR vs MR of MVP	MR of MVP increases with Valsalva and standing
PDA vs cervical venous hum	Compression of neck veins
Supraclavicular bruit vs AS	Extension of shoulder and compression of sub clavian artery increase the bruit
Small VSD vs PS	Amyl nitrite increases PS, decreases VSD Phenylephrine increases VSD, no effect on PS
Large VSD with fixed PAH vs hyperkinetic PAH	Amyl nitrite increases VSD murmur in hyperkinetic PAH
S2-OS vs S2 split (A2-P2)	Respiration effects S2 split but has no effect on S2-OS Phenylephrine decreases S2-OS Sudden standing reduces S2 split, but has no influence on S2-OS
S4-S1 vs S1 split	S1 split widens with inspiration and Passive leg raising Sudden standing shortens S1 split, decreases S4 (RV)
Ejection click of AS vs PS	In PS it is better heard during expiration (variable)
S3 or S4, LV vs RV	Inspiration increases right and expiration left sided S3/S4 Supine/passive leg raising enhances right sided S3/S4 Standing decreases right sided S3/S4
Ejection click vs non-ejection click of MVP	Standing, Valsalva enhance the non-ejection click and move it towards S1

Physiologic and pharmacological maneuvers

This cannot be overemphasized as failure to use respiration to distinguish a right sided murmur from a left sided one is similar to the failure to lateralize a hemiplegia in a patient with neurologic disease.

Accompanying features

The accompanying features in each murmur help in interpreting the murmur appropriately.

Table 22.11: Interpretation of murmurs: importance of accompanying features

<i>Auscultatory event</i>	<i>Accompanying feature</i>	<i>Significance</i>
ESM of LV outflow	Slow rising pulse Bisferiens pulse Normal pulse	Fixed LV outflow obstruction AS+AR or HOCM Aortic valve sclerosis or mild AS
EDM along LSB	Collapsing pulse Normal pulse	AR PR or mild AR
Long systolic murmur at 2nd or 3rd space	Prominent 'a' wave in JVP Sustained parasternal impulse Ejection click Absence of any of the above signs	Severe RV outflow obstruction As above Valvular PS VSD small
MDM at apex	OS Loud S1 Diastolic thrill S3 Severe AR Atrial fibrillation Pure RV impulse Biventricular impulse	MS MS MS Flow murmur due to MR or post tricuspid shunts Possible Austin-Flint MS TR VSD
Pansystolic murmur at LLSB	Large 'V' wave in JVP Forcible LV impulse Sustained LV impulse	TR MR AS
Long systolic murmur at apex	S3 S4	MR AS

Though the accompanying features are invaluable in identifying a murmur, as a student practising auscultation, one should try to evaluate an auscultatory event in isolation, without the assistance of these features. In actual practice, the accompanying features are of great value in interpreting any auscultatory sign.

23 Systolic Murmurs

EJECTION SYSTOLIC MURMURS

Mechanism

There is always a time interval between the closure of the atrioventricular valve and the opening of the semilunar valve. An ejection systolic murmur always begins a while after the first heart sound. Once ejection begins, it reaches a peak by mid-systole and hardly any ejection occurs in the late phase of systole. As a result, these murmurs start after an interval from the first heart sound, reach a peak by mid-systole and taper off by late systole. The most important members of this group are the murmurs of aortic and pulmonic stenosis. These murmurs will be dealt with in some detail.

Various causes of ejection systolic murmurs are depicted in Fig. 23.1. The location, length, character of murmur, response to various maneuvers, and associated features like an ejection click or regurgitant murmur will differentiate the various causes one from the other.

LEFT VENTRICULAR OUTFLOW OBSTRUCTION

Left ventricular outflow obstruction occurs due to a variety of causes (Table 23.1).

Even in India, isolated aortic stenosis is not rheumatic. Calcific degeneration of congenitally deformed bicuspid valve is a common cause of aortic stenosis. In rheumatic aortic stenosis, the basic tricuspid architecture of the valve is preserved. The cusps are fused along the commissures by the rheumatic process leading to a conical valvular mass with a narrow orifice in the middle.

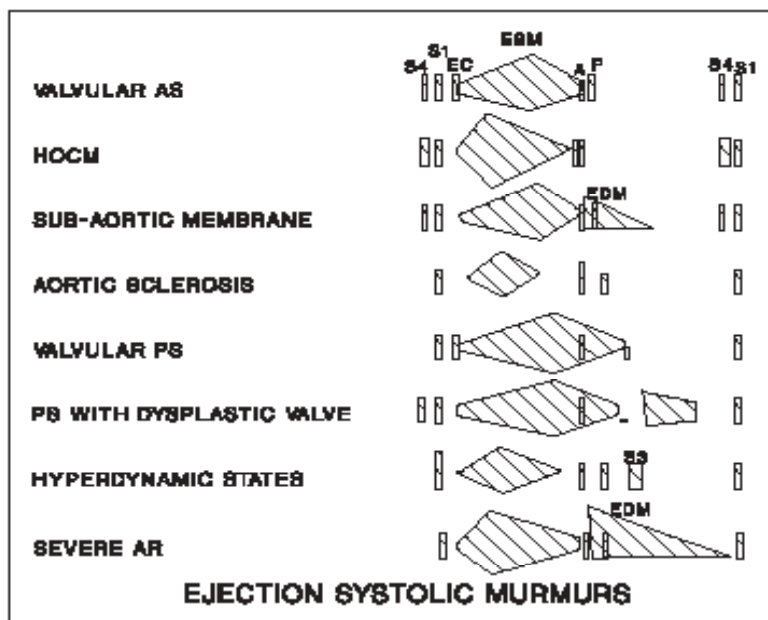


Fig. 23.1: Ejection systolic murmurs

Table 23.1: Causes of left ventricular outflow obstruction

<i>Valvular</i>	<i>Supravalvular</i>	<i>Subvalvular</i>
1. Rheumatic	1. 'Hour glass' type	1. Hypertrophic cardiomyopathy
2. Bicuspid valve	2. Diffuse type	2. Discrete membranous
3. Unicuspid valve	3. Discrete membrane	3. Tunnel type
4. Acommissural with central opening	4. Aortic dissection	
5. Unicommissural with eccentric opening	5. Homozygous hyperlipidemia	
6. Myxoid dysplasia	6. Healing aortotomy site	
7. Annular hypoplasia	7. Rubella	
8. Calcific degenerative		
9. Hyperlipidemia		
10. Fabry's disease		
11. Infective endocarditis		
12. Ochronosis		

SYSTOLIC MURMURS

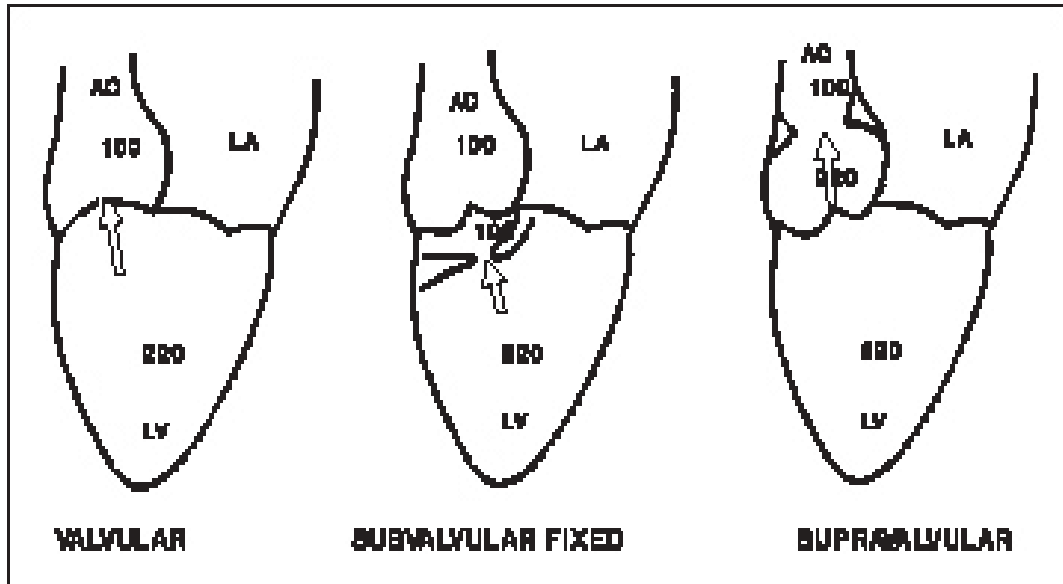


Fig. 23.2: Site of left ventricular outflow (LVOT) obstruction

The sites of obstruction can be subvalvular (fixed and dynamic), valvular and supravalvular.

The significance of each of these features will be considered in the setting of aortic stenosis. It is useful to ask the following questions when a murmur is being evaluated for aortic stenosis.

Table 23.2: Murmur of valvular aortic stenosis

Site of best audibility	Aortic area (second left space) Also heard at left sternal border, apex
Timing	Ejection systolic
Grade	Usually 3/6
Length of murmur, time of peaking	Short/medium/long
Character	Harsh
Relation to physiological act	Does not increase during inspiration Decreases on standing and straining phase of valsalva
Accompanying features	Slow rising arterial pulse Sustained left ventricular impulse Fourth heart sound, ejection click Symptoms of angina, syncope, and dyspnea

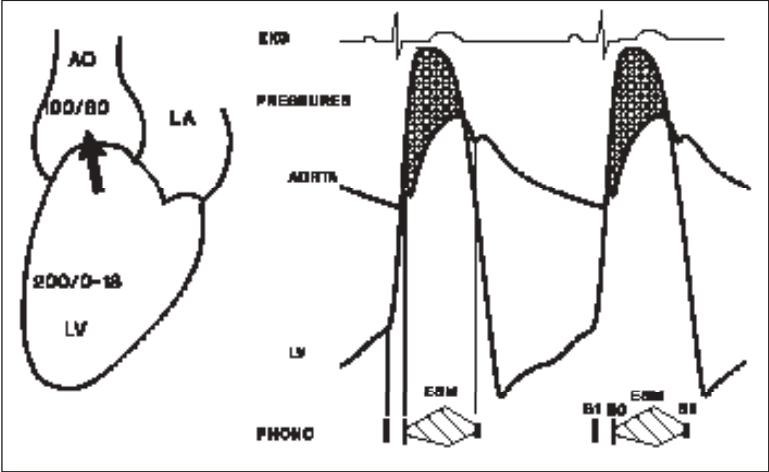


Fig. 23.3: Ejection systolic murmur of aortic stenosis

Table 23.3: Evaluation of murmur of aortic stenosis

Is it AS or one of the conditions simulating AS? Conditions simulating AS	Aortic valve sclerosis VSD MR PS
If AS, what level is the lesion? Levels of LV outflow obstruction	Valvular Subvalvular Supravalvular
If subvalvular what is the nature of obstruction? Nature of subvalvular obstruction	Dynamic (HOCM) Fixed Subvalvular membrane Fibromuscular tunnel
If fixed, what is the severity? Severity of obstruction	Mild Moderate Severe
Are there any associated defects? Associated defects in AS	AR Mitral valve disease Coarctation of aorta PDA Systemic hypertension

SYSTOLIC MURMURS

The murmur of aortic stenosis should be evaluated with the issues shown in Table 23.3 in mind.

MURMUR OF AORTIC STENOSIS

Site of best audibility/selective conduction

The murmur of valvular aortic stenosis is usually best heard at the right second space, though it is also well heard at the left sternal border and apex (Fig. 23.4).

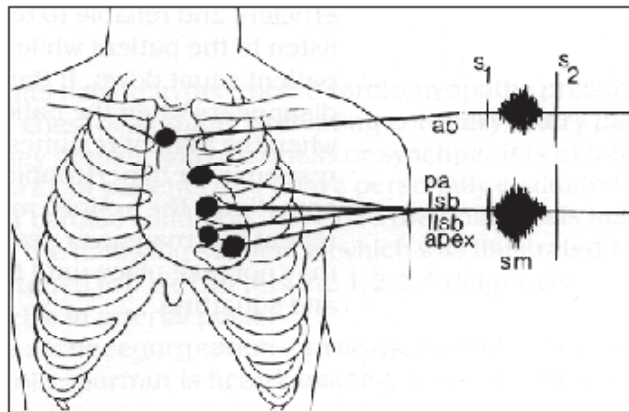


Fig. 23.4: Possible sites of best audibility of the murmur of HOCM

ao: aortic area, pa: pulmonary area, lsb: left sternal border,
llsb: lower left sternal border, sm: systolic murmur

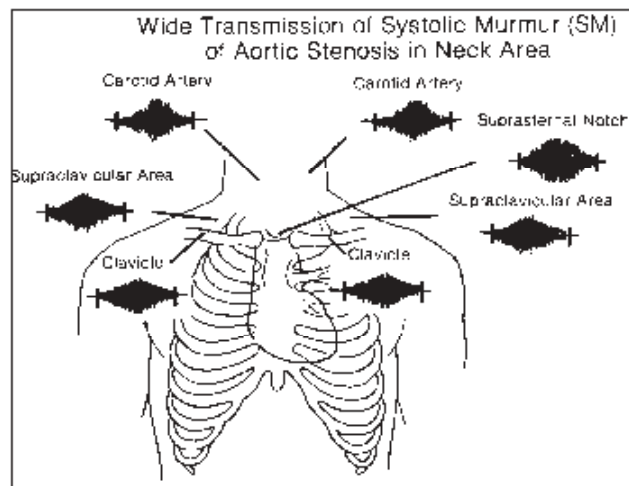


Fig. 23.5: Wide audibility of the murmur of AS

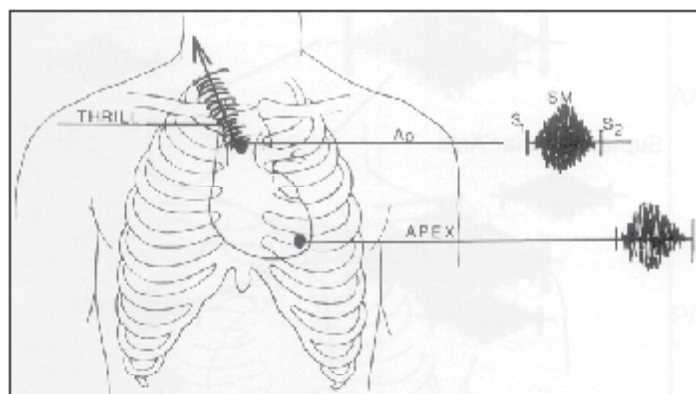


Fig. 23.6: A 60-year-old man, calcific AS; aortic stenosis grade 5/6 aortic area, harsh musical murmur at apex

It is selectively conducted to the neck vessels particularly to the right carotid (Fig. 23.5). Any deviation from this has significance. When the murmur is heard only at the apex and nowhere else, a mistaken diagnosis of mitral regurgitation is often made, but the lack of conduction to the axilla and back is helpful.

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Timing

The presence of the ejection systolic murmur localizes the lesion to the outflow from either ventricle. All other causes of ejection systolic murmur need to be distinguished from the murmur of aortic stenosis.

Table 23.4: Site of best audibility and significance in aortic stenosis

<i>Site of best audibility/ selective conduction</i>	<i>Significance</i>
Best audible at right second space, conducted to right carotid	Valvular non-calcific AS
Best audible at left sternal border, no carotid conduction	Subvalvular AS (fixed or dynamic) Calcific AS (loss of jet) Mistaken VSD Mistaken MR
Carotid murmur with or without right second space murmur	Supravalvular AS Carotid stenosis
Audible only at apex	Calcific AS in elderly with emphysema, where apex is the only part of the heart coming into contact with chest wall Mistaken for MR

Length of the murmur and time of peaking in systole

The longer the murmur and the later in systole the murmur peaks, the more severe the aortic stenosis. The duration of the murmur is a reflection of the duration of pressure difference across the valve. This correlates with the slow rising pulse and the sustained apical impulse of aortic stenosis. The duration of the murmur correlates with the severity of aortic stenosis when the cardiac output is within normal limits. Any significant change in cardiac output makes this sign unreliable. Overestimation of the severity of aortic stenosis is a feature of high cardiac output states and underestimation is a feature of low cardiac output states.

Conditions where it may be **overestimated** are:

- Anemia
- Thyrotoxicosis
- Pregnancy
- Associated aortic regurgitation
- Associated patent ductus arteriosus
- Anxiety
- Tachycardia

With a decreased cardiac output, underestimation is likely. With heart failure, the murmur decreases in intensity and length (Table 23.5). In systemic hypertension

Table 23.5: Conditions where the severity of aortic stenosis is underestimated

<i>Condition</i>	<i>Mechanism</i>
Heart failure	Low cardiac output
Polycythemia	Increased viscosity of blood
Associated proximal obstruction	Low cardiac output
	Mitral stenosis
	Tricuspid stenosis
Associated proximal regurgitation or shunt	Low cardiac output
	Mitral regurgitation
	Ventricular septal defect
Associated systemic hypertension or coarctation	Obliteration of gradient
CAD/myocardial infarction	Low cardiac output
Hypothyroidism	Low cardiac output
Elderly female	Low cardiac output
Higher NYHA class	
Atrial fibrillation	

and coarctation, as the aortic pressure increases the gradient across the valve gets obliterated and the murmur may become shorter and less intense. If the murmur is short with disproportionately severe left ventricular hypertrophy and prominent S4, hypertrophic cardiomyopathy is likely as the degree of left ventricular hypertrophy and severity of outflow obstruction may not go hand in hand in this condition. If the murmur is very short with normal upstroke of the arterial pulse and no left ventricular hypertrophy, aortic valve sclerosis is likely.

Character

The combination of low- and high-frequency components give the aortic stenosis murmur its harsh or rough quality. The rough component of the murmur is best heard at the right second space and is localized to that area, as low-frequency noises are not widely transmitted. The high-frequency soft component, however, is widely transmitted especially to the apex. This soft murmur heard at the apex simulates the murmur of mitral regurgitation closely (Gallavardin phenomenon). But the lack of conduction to the axilla and back is helpful.

Relationship with a physiological act

The useful physiological maneuvers are described below. These maneuvers have to be tried in all patients with aortic stenosis even if the diagnosis appears certain, as it permits one to gain experience of how a murmur of fixed obstruction behaves. Any deviation from this in hypertrophic cardiomyopathy is easy to recognize. There is significant variation in the response of fixed aortic stenosis to each of these maneuvers from patient to patient.

- Respiration
- Supine position
- Supine, passive leg raising
- Supine left lateral
- Sitting, leaning forward
- Standing
- Squatting
- Isometric hand grip
- Valsalva maneuver

Maneuvers like inspiration, standing, the straining phase of Valsalva and prolonged squatting by decreasing the venous return to the left ventricle, reduce

SYSTOLIC MURMURS

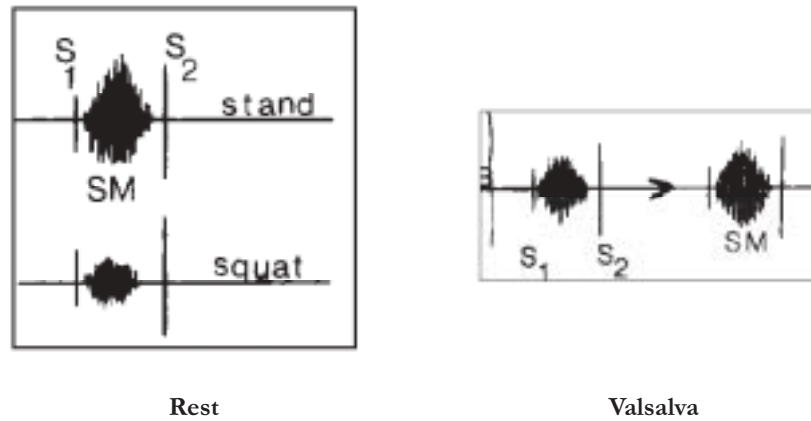


Fig. 23.7: Murmur of HOCM increasing by Valsalva and on standing

Table 23.6: Influence of various maneuvers in aortic stenosis

Maneuver	Fixed obstruction	Dynamic obstruction
Respiration	No change	May increase with inspiration
Standing	Decrease	Increase
Valsalva	Decrease	Increase
Squatting	Decrease	Increase

the cavity size and increase the obstruction in hypertrophic obstructive cardiomyopathy.

Accompanying features

Presence of symptoms such as angina, syncope and dyspnea suggest severe aortic stenosis and correlate with prolonged ejection systolic murmur with a late systolic peaking. On the other hand, when the murmur is short in the presence of these symptoms, hypertrophic cardiomyopathy or associated heart failure should be considered. In hypertrophic cardiomyopathy, the degree of outflow obstruction and symptoms are unrelated to each other.

Symptoms	Signs
Angina	Slow rising arterial pulse
Syncope	Sustained apical impulse
Dyspnea	Fourth heart sound Ejection click

The slow rising arterial pulse and a sustained apical impulse go hand in hand with a long ejection systolic murmur and a delayed peak. A normal arterial pulse is consistent with a short ejection murmur with an early peak. If the arterial pulse is normal with a long ejection murmur, murmurs simulating aortic stenosis are likely (mitral regurgitation, ventricular septal defect). If the apical impulse is not sustained with a long systolic murmur, mitral regurgitation is likely. A long systolic murmur is usually accompanied by fourth heart sound in aortic stenosis due to fixed obstruction. In dynamic obstruction due to hypertrophic cardiomyopathy (HOCM), fourth heart sound may be heard with a short murmur or no murmur, as diminished ventricular compliance is a fundamental feature in hypertrophic obstructive cardiomyopathy with or without obstruction. If fourth heart sound is not heard with a long murmur, but a third heart sound is heard, mitral regurgitation is likely. A constant ejection click distinguishes aortic stenosis from pulmonic stenosis. Again with an ejection click the ventricular septal defect or mitral regurgitation are unlikely and aortic stenosis is likely.

The ejection click localizes the obstruction to the valve and suggests a mobile non-calcific valve. Associated aortic regurgitation localizes the obstruction to the valve or subvalvular fixed obstruction and hypertrophic obstructive cardiomyopathy is unlikely.

The importance of aortic regurgitation in aortic stenosis is:

- Fixed valvular or subvalvular obstruction is likely.
- The accompanying systolic murmur is likely to be due to AS or VSD and not due to pulmonic stenosis.
- A 'new murmur of AR' in AS may mean infective endocarditis in a febrile patient with AS.

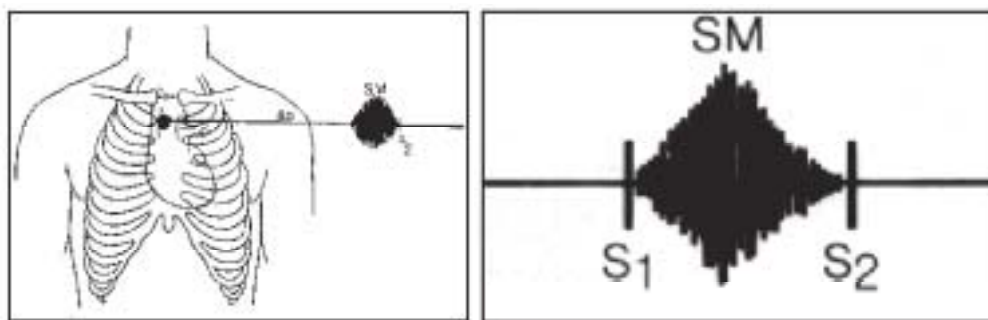


Fig. 23.8: Calcific aortic stenosis, with no ejection click. Note faint A2.

SYSTOLIC MURMURS

Table 23.7: Aortic valve sclerosis (AVS) versus aortic stenosis (AS)

<i>Feature</i>	<i>AVS</i>	<i>AS</i>
Murmur	Short/medium pitch	Long/rough
AR	Absent	May be present
A2	Normal/increased	Diminished/absent
S2	Normal split	Single/reversed split
LVH	Absent	Present
S4	Absent	May be present
Calcification	Rare	May be present
Arterial pulse	Normal	Slow rising

Once the murmur of aortic regurgitation is audible, the systolic murmur is unlikely to be related to aortic valve sclerosis (Table 23.7).

In addition to the various physiological maneuvers that may be performed to differentiate various murmurs, changes in relation to ventricular premature contractions also give clues to proper identification of murmur. Murmur of mitral regurgitation seldom changes in post-ectopic beat, while aortic stenosis murmur increases in the post-ectopic beat (Fig. 23.9).

In routine clinical practice, the clinical and electrocardiographic evidence of left ventricular hypertrophy is often used as evidence of aortic stenosis. In infants with severe aortic stenosis and heart failure, the ECG may show right axis deviation and right ventricular hypertrophy and can be misleading.

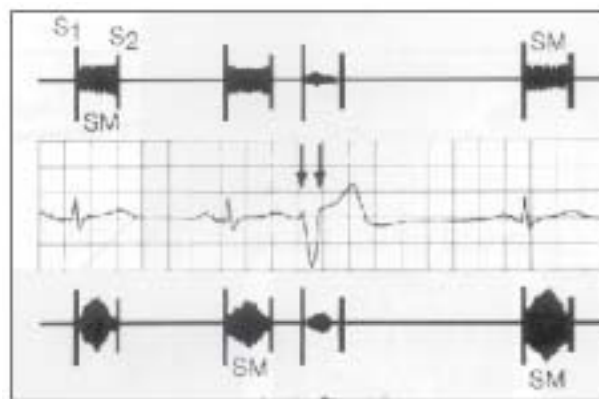


Fig. 23.9: Systolic murmur due to mitral regurgitation remains unchanged in the beat following a ventricular premature contraction, whereas the murmur increases in intensity in aortic stenosis.

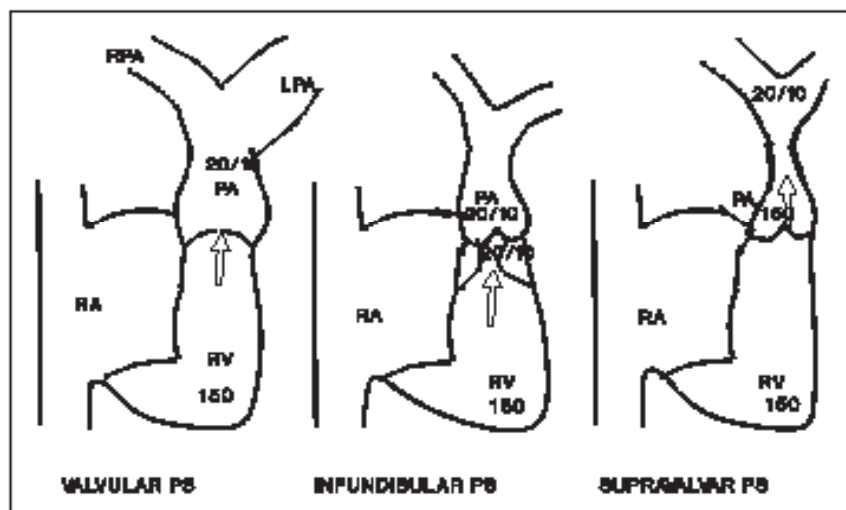
Table 23.8: Causes of RV outflow obstruction

<i>Valvular</i>	<i>Subvalvular</i>	<i>Supravalvular</i>
<i>Congenital</i> Domed (42%) Tricuspid (6%) Bicuspid (10%) Unicommissural (16%) Hypoplastic annulus (6%) Dysplastic (19%) <i>Carcinoid</i> <i>Rheumatic</i>	Congenital Infundibular stenosis Fibrous band at the junction of infundibulum and RV Fibromuscular obstruction beneath the valve Double chambered RV Neoplasm	Congenital Main trunk Right and left pulmonary arteries Tumour (external compression) Thrombus Rubella

RIGHT VENTRICULAR OUTFLOW OBSTRUCTION

Unlike aortic stenosis, the commonest cause of pulmonic stenosis is congenital. Acquired RV outflow obstruction is extremely rare (Table 23.8).

The clinical features of RVOT obstruction depend on the level of obstruction. The presence of ejection click localizes the site to the valve. Isolated infundibular pulmonic stenosis is a rare condition.

**Fig. 23.10: Various levels of right ventricular outflow tract obstructions**

SYSTOLIC MURMURS

Table 23.9: Murmur of pulmonic stenosis

<i>Feature</i>	<i>Description</i>
Site of best audibility	Pulmonary area (right second and 3rd spaces)
Timing	Ejection systolic
Grade	Grade 4/6
Length, peaking	Short, moderate, long covering A2 Peaks in mid-systole, late systole
Selective conduction	Supraclavicular, left side of neck
<i>Relationship to physiological act</i>	
Respiration	Increases with inspiration
Posture	Decreases on standing
Valsalva straining phase	Decreases or disappears
<i>Accompanying features</i>	Ejection click Wide split second heart sound Diminished pulmonic sound Right ventricular fourth heart sound Sustained left parasternal lift of RVH Systolic thrill at pulmonary area Impalpable pulmonary artery Prominent <i>a</i> wave in JV pulse Features of right heart failure
Symptoms	Shortness of breath Cyanosis/clubbing Angina Syncope Noonan's syndrome Hypertelorism
General appearance	Moon facies

The significance of each of these features will be considered in the setting of pulmonic stenosis.

Evaluation

Ask the following questions to evaluate the patient with murmur of pulmonic stenosis:

With these issues in mind, the murmur of pulmonic stenosis should be evaluated systematically.

Is it PS or a conditions simulating it?	<i>Conditions simulating PS</i> Innocent systolic murmur Straight back syndrome Atrial septal defect Ventricular septal defect Idiopathic dilatation of pulmonary artery Tetralogy of Fallot
If it is PS, what is the level of obstruction?	<i>Level of obstruction</i> Valvular Subvalvular Supravalvular
What is the severity?	<i>Severity of PS with gradient across the valve</i> Mild (<50 mmHg) Moderate (50–70 mmHg) Severe (>70 mmHg)
Is it isolated PS or part of a complex defect or syndrome?	<i>PS as a part of complex defect</i> Tetralogy of Fallot L-TGA <i>PS as part of a syndrome</i> Noonan syndrome Rubella syndrome
Is there pulmonary incompetence?	<i>Pulmonary incompetence occurs with</i>
Is it dysplastic valve?	Dysplastic valve as in Noonan's syndrome Infective endocarditis in PS Post-surgical valvotomy Post-balloon valvotomy As in Noonan's syndrome
Are there associated defects?	<i>Associated defects with PS</i> ASD VSD

MURMUR OF PULMONIC STENOSIS

Timing

The murmur of pulmonic stenosis is as a rule an ejection systolic (Fig. 23.11). A pansystolic murmur is suggestive of either a ventricular septal defect or tricuspid regurgitation. When the murmur is long, as in severe pulmonic stenosis, the ejection systolic murmur can be mistaken for the pansystolic murmur of ventricular septal defect. This confusion is common and is best resolved by the realization that there is no difficulty in distinguishing mild pulmonic stenosis from moderate or

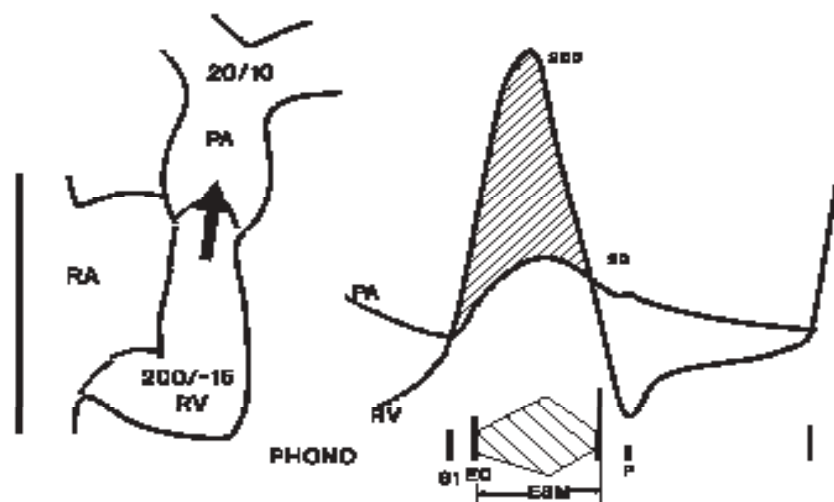


Fig. 23.11: Ejection systolic murmur of pulmonic stenosis

large ventricular septal defect. It is the small ventricular septal defect that is often confused with moderate or severe pulmonic stenosis because of the long systolic murmur which is common for both. The accompanying features of severe pulmonic stenosis, like prominent *a* wave in the neck veins, the sustained parasternal impulse, audible fourth heart sound, wide split second heart sound, and the ejection click help in this distinction. A small ventricular septal defect will have no accompanying features other than the long systolic murmur.

Site of best audibility/selective conduction

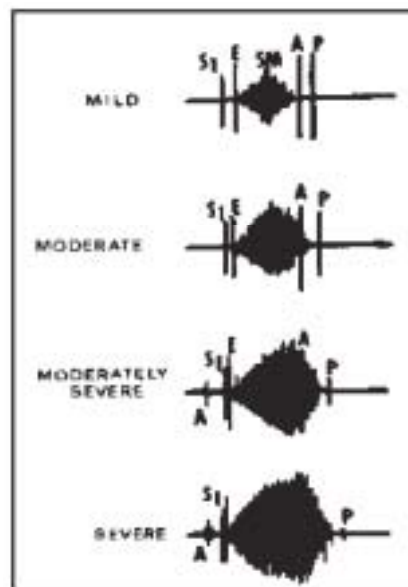
The murmur of valvular pulmonic stenosis is generally best audible at the left second or third space but is also audible at the fourth space along left sternal border. It is selectively conducted to the supraclavicular area and the left side of the neck. The murmur of ventricular septal defect is generally not conducted more to the right side of the neck than left.

The murmur of valvular pulmonic stenosis is usually best heard at the left second space but can occasionally be better audible at the third space. In other words, the murmur at the third space may be due to valvular or infundibular pulmonic stenosis (Table 23.10). The murmur of supravulvular pulmonic stenosis is better heard superiorly, along the infraclavicular region, and laterally. This murmur is usually more superficial than that of its valvular counterpart. The murmur of a double chambered right ventricle is usually best heard at the third space. The

Table 23.10: Significance of the site of best audibility

<i>Site of best audibility/ conduction</i>	<i>Significance</i>
Left 2nd space	Valvular PS
Infraclavicular and away from midline	Supravalvular PS
Left 3rd or 4th space	Infundibular PS or double chambered RV
Right 2nd or 3rd space	PS with L-TGA (PA located to right of aorta)
Conduction to left side of neck	Valvular PS
Conduction to left side of neck	Valvular PS is more likely; ventricular septal defect is less likely
Failure of conduction to left side of neck	Infundibular PS is likely

murmur of pulmonic stenosis in association with L-transposition of great arteries is best heard along the right sternal border, as the pulmonary artery is posterior and to the right of the sternum. The pulmonary ejection systolic murmur of atrial septal defect is often mistaken for pulmonic stenosis. This murmur of atrial septal defect is usually grade 3/6 or less but is widely audible all over the lung fields. This disproportion of less intense murmur at the pulmonary area with wide audibility all over the chest is suggestive of atrial septal defect.

**Fig. 23.12: Spectrum of auscultatory findings in valvular PS**

Grade, length of murmur and time of peaking in systole

The murmur of pure pulmonic stenosis is usually at least grade 4/6 (murmur with thrill). In general, the louder, longer and late peaking murmur, is associated with more severe pulmonic stenosis. In tetralogy on the other hand, these features are inversely related to the severity of pulmonic stenosis. This is related to the associated non-restrictive ventricular septal defect that permits more right to left shunt as the severity of pulmonic stenosis increases. In other words, in pure pulmonic stenosis, the right ventricle has the obligation to empty only into the pulmonary circulation; as the severity of pulmonic stenosis increases the loudness of the murmur also increases. In tetralogy the right ventricle has no obligation to empty into the pulmonary circulation, in view of the associated ventricular septal defect that allows the right ventricle to decompress into the left ventricle and aorta. The length or the loudness of the murmur is inversely related to the severity of the tetralogy. The mechanisms that influence the intensity of the murmur also influence the length of the murmur in a similar fashion. The length of the murmur is more reliable than the intensity. In adults with thick chest wall, the murmur is not particularly loud and the thrill is often absent but the length of the murmur correlates with the severity of pulmonic stenosis. In patients with right ventricular failure and associated right to left atrial shunt the forward cardiac output across the pulmonary valve decreases and the murmur decreases or may be absent. This may be mistaken for tetralogy or, surprisingly, for pulmonary arterial hypertension. The clue lies in looking for the faint murmur (grade 1 to 2/6) of pulmonic stenosis at the pulmonary area. This murmur though faint, is longer in duration and is too long for the ejection systolic murmur that can occur with pulmonary arterial hypertension.

Case summary

A 17-year-old girl was seen in the outpatient clinic for shortness of breath and cyanosis. She had moderate cyanosis and clubbing of extremities. The arterial pulse and blood pressure were normal. The JVP was elevated (8 cm above sternal angle) with a prominent 'a' wave measuring 12 cm above the sternal angle. The other pulsations were unremarkable. The apical impulse was diffuse with the point of maximal impulse of grade 2/3 and sustained with a palpable fourth heart sound. There were no thrills but pulsations were seen at the 2nd and 3rd spaces on the left. There was no palpable pulmonic sound. The first heart sound was normal, the second heart sound was single and was interpreted as a loud pulmonic sound. The right ventricular fourth heart sound was heard. There was

no click. A soft pansystolic murmur (grade 3/6) was heard along the lower left sternal border. There was grade 2/6 ejection systolic murmur at the pulmonary area. This was interpreted as the murmur related to pulmonary hypertension. The ECG showed severe right ventricular hypertrophy and right atrial enlargement. The chest X ray showed cardiac enlargement of the right atrium and ventricle with prominent main pulmonary artery and left pulmonary artery. The right pulmonary artery shadow is hidden behind the right atrial shadow. A diagnosis of:

- Severe pulmonary arterial hypertension, ? primary
- Right heart failure
- Right to left atrial shunt
- Normal sinus rhythm
- Functional class IV

was made. She was discussed in a combined cardiology conference and cardiac catheterization was decided against due to the high risk involved and also that nothing could be offered even if it were due to PAH related to Eisenmenger syndrome. While awaiting discharge, she was seen by one of the trainees who felt that the murmur at the pulmonary area, though faint, was too long to belong to pulmonary hypertension and the possibility of pulmonic stenosis existed. It was decided to catheterize her but while awaiting the procedure she died suddenly in the ward. The autopsy revealed:

- Severe valvular pulmonary stenosis (pin hole)
- Patent foramen ovale

This patient illustrates the importance of the duration of the systolic murmur of pulmonic stenosis. The pulsation in the left second or third space, single second heart sound, and the dilated main pulmonary artery can be misleading and the differential diagnosis of pulmonic stenosis involves pulmonary hypertension in certain clinical settings. Severe right ventricular hypertrophy in pulmonic stenosis when associated with severe outflow hypertrophy occasionally encroaches or displaces the main pulmonary artery posteriorly to produce pulsations in the left second or third space. The second heart sound in primary pulmonary hypertension is often single and not impressively loud unlike in pulmonary hypertension due to other conditions. The pulmonary arterial enlargement can be unimpressive in some patients with primary pulmonary hypertension. This patient was seen in 1970s when echocardiography was not available. A mistake of this nature is generally less likely but occurs even today as the following patient illustrates.

SYSTOLIC MURMURS

Case summary

A 20-year-old college girl was evaluated for dyspnea and cyanosis. After a clinical and a two-dimensional echocardiographic examination, a diagnosis of:

- Primary pulmonary hypertension
- Right to left shunt at atrial level

was made. This girl also had features similar to the described patient above, with a faint but long systolic murmur suggestive of severe pulmonic stenosis with right to left shunt at atrial level. A repeat echocardiographic examination suggested severe valvular pulmonic stenosis. At cardiac catheterization she was found to have:

- Severe valvular pulmonic stenosis
- Dynamic infundibular pulmonic stenosis
- Right ventricular failure
- Right to left atrial shunt

She underwent a percutaneous balloon valvuloplasty with an excellent outcome and is doing well an year later.

This patient illustrates (over-illustrates) the importance of the length of systolic murmur at the pulmonary area in a patient with a diagnosis of pulmonary hypertension or right heart failure, even when the diagnosis was made after an echocardiographic examination.

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Selective conduction

The murmur of pulmonic stenosis is selectively conducted to the infraclavicular region and the left side of the neck. This is related to the direction of the jet and anatomical bony contiguity. The murmur of ventricular septal defect is generally not conducted to the neck. The murmur of infundibular pulmonic stenosis and double chambered RV are usually not conducted to the neck. The pulmonic stenosis in association with D-transposition of great arteries, is poorly conducted to the infraclavicular region and is well conducted to the lung fields.

Response to various maneuvers

The murmur of pulmonic stenosis is usually increased during inspiration. This expected response is seen in only 60 per cent of patients with pulmonic stenosis and the absence of this sign is common. In the presence of severe right ventricular failure, the inspiratory increase fails to occur. The murmur decreases or disappears promptly during the straining phase of the Valsalva maneuver and reappears promptly after the release. The murmur of aortic stenosis on the other hand, decreases or disappears after a few seconds of straining and reappears after a lag

period of a few seconds. This is related to the reservoir of pulmonary vascular bed being available for the left heart.

Accompanying features

a) *The ejection click (EC)*: The EC not only distinguishes pulmonic stenosis from ventricular septal defect but also localizes the obstruction to the valve. Even with a valvular pulmonic stenosis, the click is absent in patients with dysplastic valve, absent pulmonic valve with annular stenosis or a very severe pulmonic stenosis.

Conditions with valvular pulmonic stenosis where EC may be absent:

- It may not be valvular pulmonic stenosis
- Dysplastic pulmonary valve
- Absent pulmonary valve
- Very severe pulmonic stenosis

The variability of the click with respiration (expiratory increase) distinguishes pulmonic stenosis from aortic stenosis. The click is often better appreciated one intercostal space below the site of best audibility of the murmur.

b) *The second heart sound*: The wide and variable split of the second heart sound with diminished P2 distinguishes pulmonic stenosis from ventricular septal defect. A fixed split occurs with associated atrial septal defect or right heart failure.

c) *The fourth heart sound*: Presence of the fourth heart sound makes a tetralogy unlikely and pure pulmonic stenosis likely. The gradient across the outflow is usually more than 70 mmHg when the fourth heart sound is present; the right ventricular pressures are usually suprasystemic.

d) *Prominent a wave in JVP*: This has the same significance as the fourth heart sound but is more easily and frequently appreciable than the fourth heart sound.

e) *The parasternal impulse*: A sustained parasternal impulse is consistent with pulmonic stenosis but a hyperkinetic impulse suggests atrial septal defect. Isolated parasternal impulse without LV impulse rules out ventricular septal defect as the cause of long systolic murmur.

f) *The murmur of pulmonary incompetence*: The low frequency murmur of low pressure pulmonary incompetence localizes the lesion to the valve. Pulmonary incompetence is also common with dysplastic valve or with infective endocarditis. A dysplastic valve is not easily amenable for balloon valvuloplasty.

OTHER EJECTION MURMURS AT THE BASE

EJECTION SYSTOLIC MURMUR OF AORTIC VALVE SCLEROSIS

The ejection systolic (ESM) murmur is common in the elderly and is due to thickening and occasionally mild calcification of the valve. It is short in duration, peaks early in systole, and is softer than the murmur of aortic stenosis. The second sound is normally split with normal or slight accentuation of aortic sound. This murmur needs to be distinguished from aortic stenosis (Table 23.7).

EJECTION SYSTOLIC MURMUR OF AORTIC REGURGITATION

Even in the absence of stenosis, aortic regurgitation of moderate or severe degree is often accompanied by an ejection systolic murmur due to the large stroke volume that has to be moved across the aortic valve. This murmur may be grade 4/6 or more and may be confused for an associated aortic stenosis with aortic regurgitation (Table 23.11).

When the diastolic pressure is less than 40 mmHg, free severe aortic regurgitation is likely, and associated aortic stenosis is unlikely. Systolic thrill over the precordium is not a feature with the ejection systolic murmur of aortic regurgitation.

EJECTION SYSTOLIC MURMUR IN THE AORTIC AREA IN THE YOUNG

Unlike in the elderly, an ejection systolic murmur in the aortic area in a young person should be considered abnormal even if the murmur is less than grade 3/6. Once aortic stenosis is ruled out, one must look for a hyperkinetic state or an underlying aortic regurgitation. The early diastolic murmur of aortic regurgitation is more difficult to hear than the systolic counterpart. It is in this setting, that all

Table 23.11: Ejection systolic murmur of aortic regurgitation versus aortic regurgitation with aortic stenosis

<i>Feature</i>	<i>ESM in AS + AR</i>	<i>ESM of pure AR</i>
Systolic thrill	Precordium + carotid	Carotid thrill may occur No precordial thrill
Murmur length	Long	Short
Time of peaking	Later in systole	Early in systole

the maneuvers to bring out the murmur have to be tried. In the setting of rheumatic heart disease, with mitral and tricuspid valve disease, even a faint 1/6 ejection systolic murmur at the aortic area suggests associated aortic stenosis. This is related to the reduction in cardiac output due to two proximal lesions. At cardiac catheterization, even as low a gradient as 30 mmHg, is considered suggestive of severe aortic stenosis. The diagnostic possibilities are:

- Underlying aortic regurgitation
- Mild aortic stenosis
- Aortic stenosis in association with tight mitral stenosis
- Congenital bicuspid aortic valve
- All conditions with aortic run-off
- Hyperkinetic circulatory states
- Hyperkinetic heart syndrome

Case summary

A 16-year-old boy presented with palpitation and weakness of 2 months duration. Earlier he was seen at a hospital in another city where he was evaluated by cardiac catheterization and left ventricular angiography but an echocardiogram was not done. The intracardiac pressures and flows were found to be normal and the left ventriculogram showed a normally contracting left ventricle. The cardiovascular system was pronounced normal with an 'innocent systolic murmur' of aortic origin. Physical examination revealed an otherwise normal cardiovascular system but for a short ejection systolic murmur of grade 2/6 at the aortic area. A short early diastolic murmur was heard along the left sternal border on prompt squatting and later was also heard with held expiration. A diagnosis of mild aortic incompetence was made. The patient was advised normal activity and prophylaxis for rheumatic fever and infective endocarditis.

This patient illustrates the importance of 'unexplained' systolic murmur at the aortic area in the young. Additionally, even at cardiac catheterization, aortic regurgitation was missed because selective aortic root angiogram was not done as aortic regurgitation was unsuspected prior to cardiac catheterization. The value of proper clinical evaluation as a guide to investigation is to be noted.

ESM lower than grade 3/6 at pulmonary area

This is the commonest of murmurs in an otherwise normal cardiovascular system. A systematic approach to this physical sign often helps in the diagnosis of conditions of non-cardiovascular origin. Various possibilities are:

SYSTOLIC MURMURS

- Innocent systolic murmur
- Pectus excavatum
- Straight back syndrome
- Kyphoscoliosis
- Pulmonary fibrosis
- Anemia
- Pregnancy
- Thyrotoxicosis
- Atrial septal defect
- Mild pulmonic stenosis
- Idiopathic dilatation of pulmonary artery
- Pulmonary arterial hypertension
- Hypertrophic obstructive cardiomyopathy
- Pulmonary AV fistula (increased flow)
- Systemic AV fistula

As the possibilities are manifold, a systematic approach is pertinent.

a) Look for anemia, thyrotoxicosis, pregnancy and AV fistula: As a first step, look for pallor over the tongue and mucus membranes to rule out anemia. Look at the thyroid gland for any enlargement or other features of thyrotoxicosis. A venous hum is often a clue to either of these conditions. Absence of pallor does not always rule out anemia. Reliable hemoglobin estimation is mandatory for all patients who visit a physician or cardiologist for cardiovascular evaluation. In all female patients, recheck menstrual history to rule out early pregnancy. All radiological investigations should be deferred until early pregnancy is ruled out. A systemic arteriovenous fistula may exhibit an ejection systolic murmur in the pulmonary area. Looking for an AV fistula involves auscultation over the liver, skull or any other site of past trauma, needle puncture or surgery. In all patients who underwent cardiac catheterization from the femoral site, the groin should be auscultated for a continuous murmur.

b) Look for chest deformity: Look for kyphoscoliosis, pectus excavatum or straight back syndrome. The last entity is often missed and is best detected by making the patient stand up and looking for loss of thoracic lordosis. A lateral view of the chest is confirmatory.

c) Others: Other conditions can be differentiated by the alterations in second heart sound, presence or absence of additional sounds, and specific chamber enlargement. It is often not realized that hypertrophic obstructive cardiomyopathy can simulate an atrial septal defect by the systolic murmur and 'reversed wide split'.

If an ejection systolic murmur develops for the first time in patients with coronary artery disease receiving long term aspirin, significant anemia due to gastrointestinal bleeding should be considered.

Innocent systolic murmurs are recognized by the company they keep, namely the normal splitting of S₂, and are often accompanied by physiological S₃ in children.

ESM lower than 3/6 at pulmonary area in coronary artery disease

This could have significance in

- Anemia of gastrointestinal origin
- Peptic ulcer
- Aspirin induced peptic ulceration

<i>Normal split of second heart sound, P2 normal or mild accentuation</i>	
Look for pallor	Functional murmur of anemia
Look for thyroid enlargement/signs of thyrotoxicosis	Functional murmur
Last menstrual period/early pregnancy	Functional murmur
Chest deformity	Functional murmur
Continuous murmur over any site of AV fistula, high volume pulse	Functional murmur
<i>Abnormal split of second heart sound, with additional sounds</i>	
Wide 'fixed' split S ₂ , P2 normal or louder, tricuspid MDM	Atrial septal defect
Wide, variable split, decreased P2, EC	Mild pulmonic stenosis
Wide variable split, P2 normal, no tricuspid MDM	Idiopathic dilatation of pulmonary artery
Close split or single S ₂ , increased P2	Pulmonary arterial hypertension
Reversed split, LV S ₄ , LVH	Hypertrophic obstructive cardiomyopathy

SYSTOLIC MURMURS

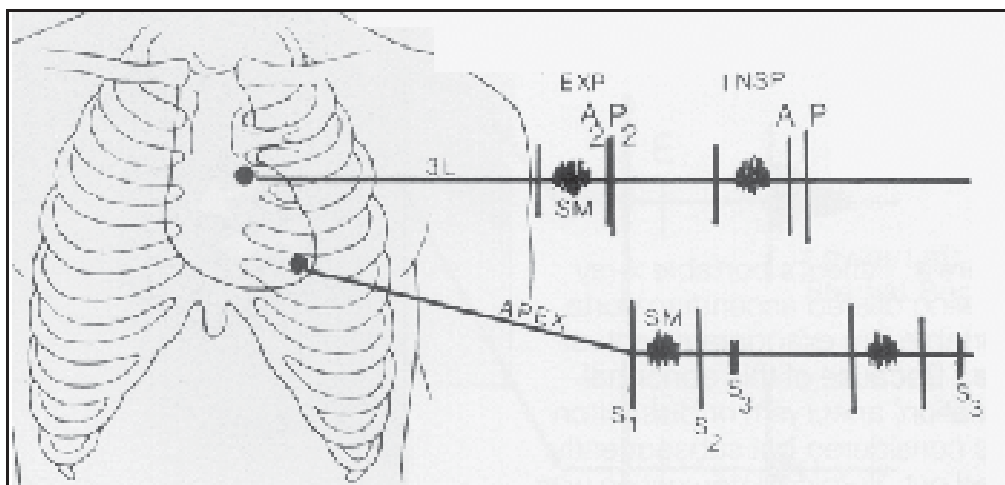


Fig. 23.13: Innocent systolic murmur, normal S2 split, physiological S3

- Malignancy of GI tract
- Coronary AV fistula to RA or RV
- Left main coronary arising from pulmonary artery (ALCAPA)
- Hypertrophic cardiomyopathy mistaken for CAD

Surprisingly, many patients do not notice malena for long periods. A 50-year-old man had angina class 2, with angiographic mid left anterior descending lesion of 75 per cent and normal LV function. He was asymptomatic with drugs and was on medical follow up. Three months later, he came with increasing angina with occasional rest angina. Physical examination was otherwise unremarkable but for an ejection systolic murmur 3/6 at the pulmonary area. A repeat angiogram revealed findings exactly similar to the previous one. The intracardiac pressures and LV function were normal. Review of his case revealed that he had a loud venous hum in addition to ejection systolic murmur 3/6 at the pulmonary area. His laboratory test was repeated and his hemoglobin was found to be 6.5 g% only. Upper GI endoscopy revealed an active duodenal ulcer and he was treated for it; there was prompt recovery. Aspirin was discontinued and iron was supplemented. This patient's example should not be used to mean that the best way to detect anemia is to wait for an ejection systolic murmur at the pulmonary area. It should be looked for in the long term follow up of all patients who receive long term oral anticoagulants, aspirin or other analgesics.

PANSYSTOLIC MURMURS

A pansystolic murmur by definition begins with the first heart sound and occupies all of the systole up to the second sound on its side of origin. The configuration of the murmur is plateau or even. A pansystolic murmur means a pansystolic pressure difference between the two chambers. This pressure relationship exists with left ventricle and left atrium as in mitral regurgitation, right ventricle and right atrium as in tricuspid regurgitation and between the two ventricles as in ventricular septal defect. Nowhere else in the heart does a pansystolic pressure difference exist to permit a pansystolic murmur. The murmurs of mitral regurgitation, tricuspid regurgitation and ventricular septal defect will be described.

MITRAL REGURGITATION

The competence of the normal mitral valve is dependent on the function of the following structures (Fig. 23.14).

- Leaflets/commissures
- Chordae
- Papillary muscles
- Left ventricle
- Mitral annulus
- Left atrium

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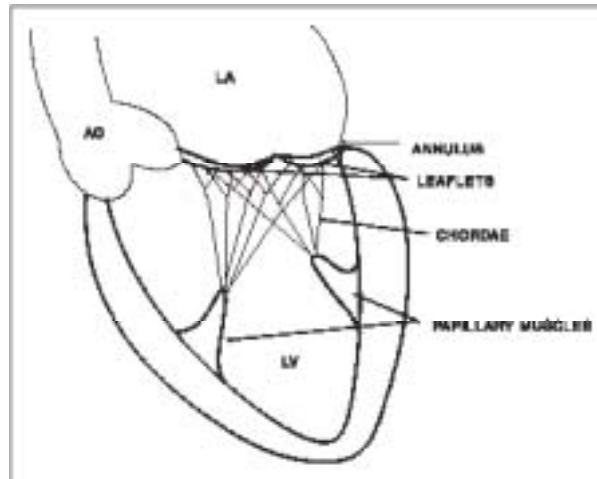
Abnormal functioning of any of the above structures can result in mitral regurgitation. The features of the murmur of mitral regurgitation should be described in full as each one of them have significance in the final assessment of the patient.

The mitral valve apparatus consists of both anterior and posterior leaflets, the chordae supporting the leaflets in systole, the papillary muscles that prevent prolapse of the leaflets due to tension applied to the chordae, the mitral annulus, the left ventricle (LV) and the left atrium (LA).

When the mitral leaflets become incompetent, blood regurgitates into the left atrium with a high pressure difference starting with the onset of systole and continuing to the end of systole. As a result, the V wave in the left atrium markedly increases.

The commonest cause of mitral regurgitation is rheumatic heart disease.

SYSTOLIC MURMURS



During the acute phase of the rheumatic process due to acute carditis, annular
Fig. 23.14: Components of mitral valve apparatus

dilatation is the principal mechanism and the leaflets show edema with normal chordae. In chronic rheumatic heart disease, progressive leaflet thickening with retraction of the cusps occurs. The posterior cusp is involved to a greater extent; as a result, it is retracted and rolled with shortening of chordae. The anterior leaflet is less thickened and the major chordae are frequently elongated, encouraging prolapse. The posterior chordae may also elongate and may rupture. Annular dilatation slowly increases and results in progressive mitral incompetence.

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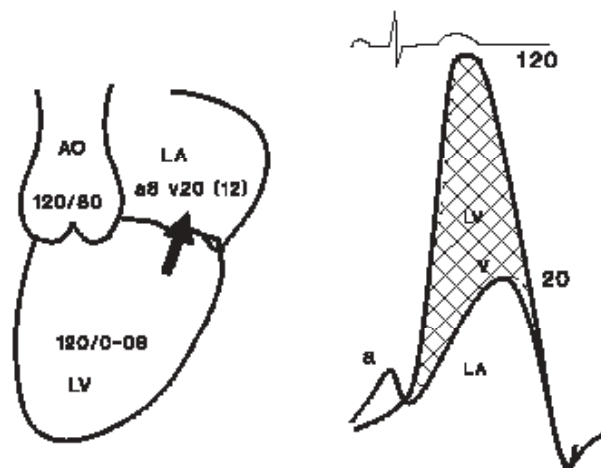


Fig. 23.15: Mechanism of pansystolic murmur of mitral regurgitation (MR)

Table 23.12: Profile of the murmur of mitral regurgitation

Site of best audibility	Apex, left sternal border
Timing	Pansystolic, late systolic, early systolic
Grading/thrill	Grade 3/6, thrill uncommon
Character	Soft, blowing, musical, honking
Selective conduction	Left axilla, back, left sternal border/aortic area
<i>Relation to maneuvers</i>	
Respiration	Fails to increase during inspiration
Posture	
Standing	No change in rheumatic MR Increases in MVP
Valsalva straining	Decrease in Rheumatic MR Increase in MVP
Cycle length	No change in rheumatic MR Change in murmur in MVP
Associated features	Hyperkinetic left ventricular impulse Diminished first heart sound Wide, variable second heart sound Third heart sound Mitral diastolic murmur Non-ejection click(s) Fourth heart sound

Timing

The murmur of mitral regurgitation is classically pansystolic and this is true in most patients with rheumatic mitral regurgitation. Late systolic murmurs occur in mitral valve prolapse and papillary muscle dysfunction. The murmur of acute mitral regurgitation is early systolic as the pressure difference between the left ventricle and left atrium gets obliterated by late systole.

The causes of non-pansystolic murmurs in mitral regurgitation are:

- Mitral valve prolapse
- Papillary muscle dysfunction
- Acute mitral regurgitation (early systolic)
- Trivial or mild mitral regurgitation (even rheumatic)
- 'Swing' mitral regurgitation

SYSTOLIC MURMURS

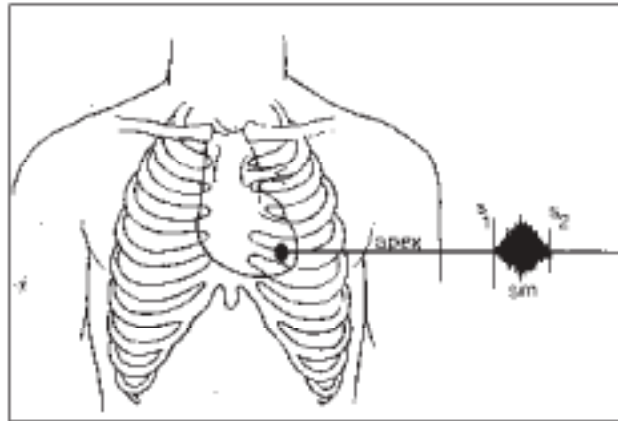


Fig. 23.16: Ruptured chordae tendinae – pansystolic murmur with mid-systolic accentuation

In acute mitral regurgitation due to papillary muscle dysfunction/rupture as in myocardial infarction, the mitral regurgitation may be faint or even silent. This is due to the accompanying left ventricular dysfunction and hypotension. In prosthetic mitral regurgitation the mitral regurgitation occurs around the valve ring (paraprosthetic) and is often silent.

The causes of silent MR are:

- All causes of acute mitral regurgitation
 - Infective endocarditis
 - Acute myocardial infarction
 - Postsurgical after closed or open commissurotomy
 - Post-balloon valvuloplasty
 - Trauma

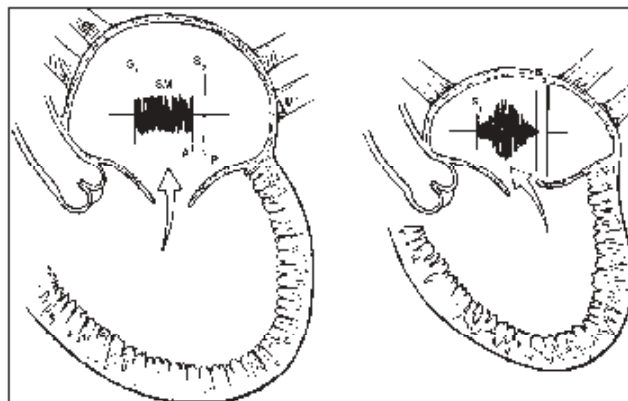


Fig. 23.17: Mitral regurgitation – chronic (left) and acute (right)

- Para-prosthetic regurgitation
- Trivial mitral regurgitation due to any cause

In trivial mitral regurgitation due to any cause, the murmur is clinically inaudible but is often detected by Doppler echocardiography. This type of mitral regurgitation has no influence over the selection of outcome of patients of mitral stenosis for commissurotomy or balloon valvuloplasty.

Site of best audibility

Rheumatic mitral regurgitation is usually best audible at the apex. In children on the other hand, the murmur is sometimes best audible at the lower left sternal border. The murmur of mitral valve prolapse is occasionally best audible at the lower left sternal border and is possibly suggestive of posterior leaflet involvement.

Grade and presence or absence of thrill

The murmur of mitral regurgitation is usually grade 3/6 and the systolic thrill is less common because the murmur is soft and of high frequency. Presence of a systolic thrill usually suggests a chordal rupture, infective endocarditis with vegetations or the systolic murmur of aortic stenosis or ventricular septal defect mistaken for mitral regurgitation.

Though systolic thrill does not negate the diagnosis of mitral regurgitation, it is useful to consider the above possibilities before ascribing it to mitral regurgitation.

Character

The murmur of mitral regurgitation is characteristically soft and blowing. Musical murmurs occur when there is an unusually vibrating structure in the pathway of regurgitation, as in ruptured chordae or the vegetations in infective endocarditis. The murmur of mitral valve prolapse is different in character from rheumatic mitral regurgitation and is usually rasping in quality. A musical honking murmur is characteristic of mitral valve prolapse. Medium frequency or harsh murmurs of mitral regurgitation are usually non-rheumatic and are generally due to mitral valve prolapse. In general, if the murmur is rough in quality, it is useful to rule out aortic stenosis or ventricular septal defect simulating mitral regurgitation.

Selective conduction

The murmur of rheumatic mitral regurgitation is most commonly conducted

selectively to the left axilla and back. This is related to the direction of the jet, which is generally directed to the left and posteriorly. In some patients with mitral valve prolapse, the murmur is selectively also conducted along the left sternal border to the aortic area. The underlying mechanism is that the jet of mitral regurgitation is directed medially along the aortic root. This type of jet is related to the dominant involvement of posterior leaflet of mitral valve in mitral valve prolapse. This type of murmur of mitral valve prolapse is often confused for aortic stenosis. However, the pansystolic timing of the murmur, the hyperkinetic apical impulse, and a normal upstroke of the arterial pulse favour mitral regurgitation. This type of murmur in mitral valve prolapse with a posterior leaflet involvement is easily amenable to repair of the valve rather than replacement. Rarely, the jet of mitral regurgitation can be so unusually directed in mitral regurgitation due to mitral valve prolapse that the murmur is selectively conducted to the right axilla. The direction of the jet in this case is directed to the interatrial septum. In children with severe mitral regurgitation, the murmur is heard well even at the pulmonary area simulating a ventricular septal defect. The basis for this is possibly the jet of mitral regurgitation reaching the area of left atrial appendage which is closely related to the pulmonary artery.

Maneuvers

Respiration: Contrary to common teaching and belief, the murmur of mitral regurgitation does not increase with expiration. More importantly, it fails to increase with inspiration, unlike its counterpart at the tricuspid valve.

Posture: The rheumatic mitral regurgitation decreases on standing or may not change significantly with posture. The tricuspid regurgitation murmur decreases with standing and increases with supine position. The mitral regurgitation of mitral valve prolapse increases on standing (smaller left ventricle) and decreases with supine position (larger left ventricle).

Valsalva: The mitral regurgitation of mitral valve prolapse increases during the straining phase of Valsalva whereas rheumatic mitral regurgitation decreases or disappears.

Phenylephrine: Decreases the mitral regurgitation of mitral valve prolapse due to reflex bradycardia and larger left ventricular cavity.

In certain situations, the murmur of MR is not obvious and requires to be carefully sought. This is more likely when the regurgitation is too mild or is due to non-rheumatic causes.

Significant MR ($> 1+$) in the setting of acute myocardial infarction is associated with a five fold increased risk of death and is a function of more extensive infarction, failure to reperfuse, previous infarction, and concurrent multivessel disease.

After each dilatation during PBMV, one should carefully listen for the murmur of MR. This murmur is often faint and non-pansystolic, and is often missed by cursory auscultation. Even a faint murmur should be given importance (Table 23.13). Other useful clues are non-ejection click audible after dilatation of the valve and a clearly palpable left ventricular impulse. The non-ejection click is due to chordal rupture. Ideally the person looking for MR should have auscultated the patient prior to balloon dilatation. As many auscultators are unreliable, Doppler echocardiographic evaluation in the catheterization laboratory is recommended.

TRICUSPID REGURGITATION

The commonest cause of tricuspid regurgitation is secondary to high right ventricular pressures either due to pulmonary arterial hypertension or pulmonic stenosis or dilatation of the right ventricle due to ventricular failure (Fig. 23.18).

Table 23.13: Searching for mitral regurgitation – situations and significance

<i>Circumstance</i>	<i>Significance</i>
Any fever of any duration	Infective endocarditis
Child suspected of rheumatic fever	Acute carditis
All patients with MS	Associated MS modifies the intervention Enhanced risk of infective endocarditis
Acute chest pain	Ischemic MR MVP
Acute MI	Ischemic MR Dilated LV
During PBMV	Any degree of MR appearing during the procedure is a contraindication to further dilatation of the valve

SYSTOLIC MURMURS

The concept of tricuspid valve complex and underlying pathophysiology is useful in understanding tricuspid regurgitation.

Right atrium

- Atrial volume
- Atrial pressure
- Atrial compliance
- Annulus

Tricuspid valve (three leaflets)

- Subvalvular apparatus
- 25 chordae (five types)
- Three major papillary muscles, several minor papillary muscles

Right ventricle

- RV volume
- RV pressure
- RV compliance
- RV architecture

Derangement of any of these components in isolation, or more often in combination, results in tricuspid regurgitation.

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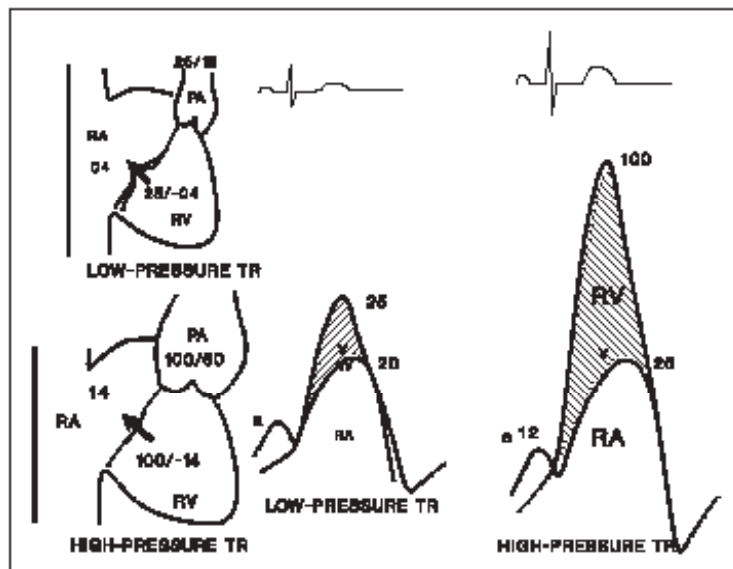


Fig. 23.18: Mechanisms of tricuspid regurgitation

Table 23.14: Profile of tricuspid regurgitation

Site of best audibility	Tricuspid area
Timing	Pansystolic
Grading	< 3/6
Character	Soft and blowing
Selective conduction	No selective conduction but is often heard to the right of sternum
<i>Relation to maneuvers</i>	
Respiration	Increases during inspiration and decreases with expiration (Carvallo's sign)
Posture	Increases with supine passive leg raising, decreases with standing
Valsalva straining	Disappears
<i>Accompanying features</i>	Signs of pulmonary hypertension Hyperkinetic right ventricular impulse Prominent <i>v</i> wave in jugular venous pulse Rapid <i>y</i> descent in jugular venous pulse Right ventricular third heart sound

Tricuspid regurgitation may result from organic tricuspid valve involvement or functional impairment due to raised right ventricle pressure. In the former, called low pressure tricuspid regurgitation, the murmur is short as there is equalization of pressures between the right atrium and right ventricle during late systole. In high-pressure tricuspid regurgitation, the murmur tends to be of high frequency and holosystolic due to high pressure gradient between right ventricle and right atrium. The importance of each of these features is detailed below.

Site of best audibility

The murmur of tricuspid regurgitation is best heard at the tricuspid area (left 4th space) and is generally not selectively conducted. It may be heard to the right of the sternum or over the liver. Very rarely, conduction into the neck veins may occur – when it can be mistaken for an aortic murmur. In Ebstein's anomaly, the murmur may be best heard laterally at the apex due to displacement of the tricuspid valve. In severe tricuspid regurgitation with enlarged right ventricle forming the apex, the murmur may be audible at the apex and even in the axilla. In this setting it may be mistaken for mitral regurgitation.

Timing and character

The typical murmur of tricuspid regurgitation is pansystolic and is indicative of tricuspid regurgitation with high right ventricle pressures. The duration of the murmur is indicative of the duration of pressure difference between the right ventricle and right atrium in systole. Non-pansystolic murmurs occur with organic tricuspid valve disease and normal right ventricle pressures. Even with high right ventricle pressures, a mild or trivial tricuspid regurgitation may be non-pansystolic. The high frequency murmur is suggestive of high pressures in the right ventricle. With normal right ventricle pressures, the murmur is lower in frequency.

Causes of short murmur

- Organic tricuspid valve disease without pulmonary hypertension
 - Rheumatic
 - Carcinoid syndrome
 - Tricuspid valve prolapse
- Acute tricuspid regurgitation
 - Infective endocarditis
 - Traumatic
 - Following catheter manipulation in right heart
 - Surgical
 - Pacemaker wire
 - Balloon dilatation
 - Blunt trauma to chest
- Mild or trivial regurgitation even with pulmonary hypertension

The higher the frequency and longer the murmur, the higher the right ventricle pressure. With high right ventricle pressures, a mild tricuspid regurgitation may produce a shorter murmur but the frequency is higher. In actual practice, the frequency is the more reliable indicator of pressures than the length of a murmur.

Grading

As a rule, the murmur of tricuspid regurgitation is less than grade 3/6 and a thrill is extremely rare. Systolic thrill in tricuspid regurgitation should raise the possibility of ventricular septal defect mistaken for tricuspid regurgitation or organic tricuspid regurgitation. Even a functional tricuspid regurgitation when severe may be associated with a thrill. Such murmurs of tricuspid regurgitation in severe

pulmonary arterial hypertension and mitral stenosis generally indicate the need for tricuspid annuloplasty in addition to the corrective surgery for mitral stenosis.

Selective conduction

The murmur of tricuspid regurgitation usually has no selective conduction, but may rarely be conducted selectively toward the right sternal border or even into the neck. The cervical conduction is related to the jet of tricuspid regurgitation directed into the superior vena cava and can be confirmed by Doppler. The cervical component of the murmur disappears by compression over the base of the neck. When tricuspid regurgitation is very severe with extreme right ventricle enlargement, the right ventricle may form the apex and the murmur may be audible at the apex or even in the axilla simulating mitral regurgitation. The inspiratory increase in murmur and pure right ventricle enlargement with apical retraction favor tricuspid regurgitation.

Relation to physiological act

Respiration: The murmur of tricuspid regurgitation increases or is heard only during inspiration, and decreases or even disappears during expiration. This is related to inspiratory increase in venous return augmenting right ventricle output (Fig. 23.19).

Failure of the murmur to increase during inspiration may mean that the murmur is probably due to an other condition, like ventricular septal defect or mitral regurgitation. Also, the murmur of tricuspid regurgitation fails to increase

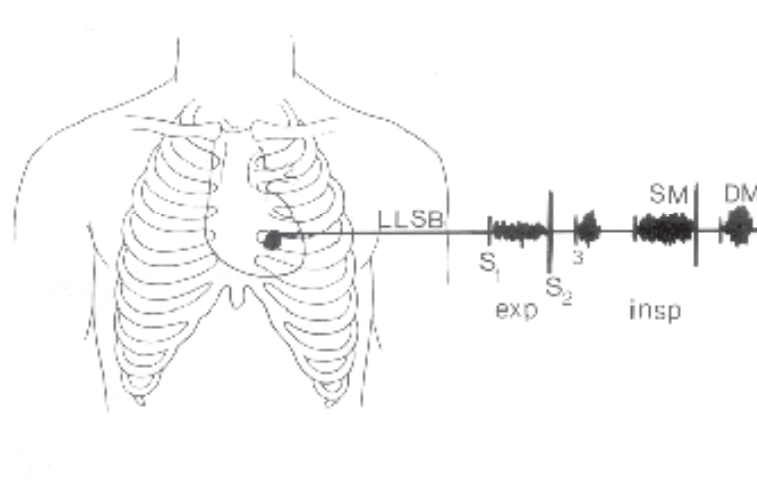


Fig. 23.19: Respiratory variation of tricuspid regurgitation murmur

during inspiration when the tricuspid regurgitation is accompanied by organic tricuspid stenosis or severe right ventricle failure. In the presence of severe right ventricle failure, the right ventricle fails to take up the challenge of additional venous return of inspiration, and fails to increase the output and thereby the murmur. The associated tricuspid stenosis prevents any further increase in venous return into the right ventricle and the tricuspid regurgitation murmur fails to increase during inspiration, though the murmur of tricuspid stenosis increases during inspiration.

In a clinical setting of severe tricuspid regurgitation and right ventricle failure with the right ventricle forming the apex, the murmur of tricuspid regurgitation may be heard at the apex and may not increase during inspiration. As a result the murmur of tricuspid regurgitation is mistaken for mitral regurgitation. This mistake is most common in pure mitral stenosis with severe pulmonary arterial hypertension and severe tricuspid regurgitation. A diffuse apical impulse with a point of maximal impulse along the left sternal border and apical retraction due to right ventricle volume load rules out mitral regurgitation of any significance.

Accompanying features

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- a) ***Signs of severe pulmonary arterial hypertension:*** The functional murmur of tricuspid regurgitation is almost always accompanied by significant pulmonary arterial hypertension. The right ventricle systolic pressure is at least 70 mmHg when high frequency and pansystolic murmur of tricuspid regurgitation occurs. In the absence of pulmonary arterial hypertension or only mild pulmonary arterial hypertension, the tricuspid regurgitation is likely to be organic.
- b) ***Sustained, hyperkinetic parasternal impulse:*** The parasternal impulse is often grade 3/3 with increased duration of impulse as the right ventricle is both volume and pressure loaded. A hyperkinetic impulse without increased duration is suggestive of normal pressure or organic tricuspid regurgitation. Pure right ventricle enlargement with a pansystolic murmur is suggestive of tricuspid regurgitation; it is unlikely to be ventricular septal defect.
- c) ***Right ventricular third heart sound:*** Almost all the patients with tricuspid regurgitation have right ventricle failure and the rapid filling of right ventricle results in right ventricle third heart sound. This third heart sound rules out associated tricuspid stenosis.

d) **Tricuspid diastolic murmur:** A tricuspid diastolic murmur increasing on inspiration suggests associated tricuspid stenosis. In severe functional tricuspid regurgitation, a tricuspid diastolic murmur may occur even in the absence of tricuspid stenosis due to large flow. This murmur does not increase significantly with inspiration.

e) **Prominent *v* wave:** The *v* wave in JVP is prominent with significant tricuspid regurgitation. However, lack of this sign does not rule out tricuspid regurgitation as significant tricuspid regurgitation can occur with little or no *v* wave in the neck.

f) **Rapid *y* descent:** Pure tricuspid regurgitation is associated with rapid *y* descent in the absence of associated tricuspid stenosis.

VENTRICULAR SEPTAL DEFECT

The ventricular septal defect is a hole or multiple holes in the ventricular septum. It may be primary, part of a complex defect, or acquired.

Classification

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Setting

- Primary or isolated
- Part of a complex defect
 - Tetralogy of Fallot
 - Complete AV canal defect
 - Corrected transposition of great arteries
 - Truncus arteriosus
 - Tricuspid atresia
 - Sinus of Valsalva aneurysm
 - D-transposition of great arteries

Size

Small VSD

- VSD restrictive and resistance index is greater than 20 units/m² BSA
- Normal RV systolic pressure
- Qp/Qs is less than 1.75

Moderate VSD

- Restrictive VSD but may raise RV systolic pressure to approximately half of LV pressure

SYSTOLIC MURMURS

- Qp/Qs may reach 2 : 1

Large VSD

- The size of aortic orifice or larger
- No resistance to flow
- RV systolic pressure approximates that of LV pressure

Location

- Perimembranous (includes LV-RA defect)
- Subpulmonary
- Below the septal leaflet of tricuspid valve
- Muscular
- With straddling or overriding of tricuspid valve

The typical murmur of ventricular septal defect is pansystolic because there is a pansystolic pressure difference between the two ventricles in communication (Table 23.15).

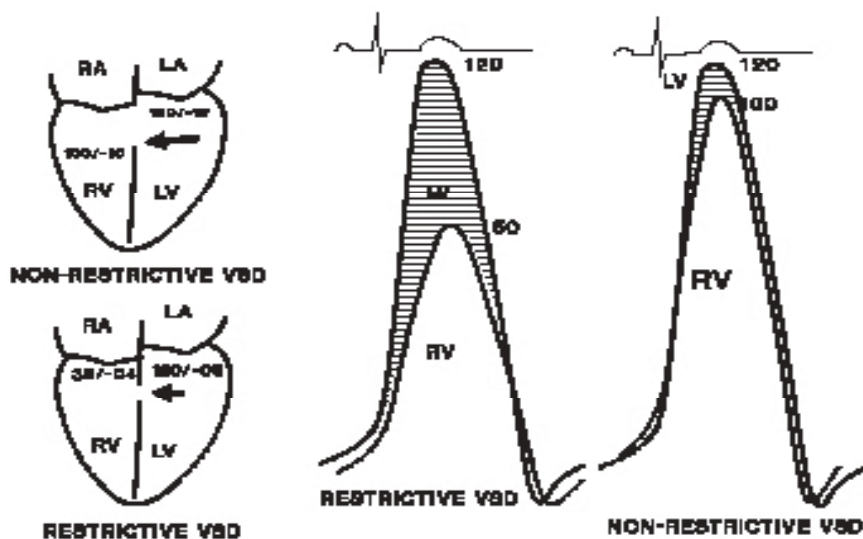
Table 23.15: Profile of the murmur of ventricular septal defect

Timing	Pansystolic
Site of best audibility	Left sternal border 2 nd to 4 th space
Grade	Grade 4/6
Character	Rough/harsh
Selective conduction	No selective conduction
<i>Relation to physiologic act</i>	
Respiration	Fails to increase with inspiration
Valsalva straining	Increases with expiration
Amyl nitrite	Decreases or disappears
Phenylephrine	Decreases or increases
<i>Accompanying features</i>	
	Biventricular enlargement
	Signs of pulmonary arterial hypertension
	Mid-diastolic murmur at apex
	Second sound normally split or single
<i>Associated lesions</i>	
	Early diastolic murmur of aortic regurgitation
	Continuous murmur of PDA
Age at which murmur is detected	Most commonly 2–6 weeks after birth

Table 23.16: Size of ventricular septal defect: definitions

<i>Restrictive defects</i> (Diameter < 1 cm/m ² BSA or orificial area < 0.8 cm ² /m ²)	RV pressure is normal Left to right shunt is $< 1.5:1$ No cardiac enlargement
<i>Small ventricular septal defect</i>	Pansystolic murmur No MDM at apex
<i>Moderate ventricular septal defect</i>	Elevation of RV pressure to less than 75% of systemic pressure Left to right shunt is $> 2:1$ Pansystolic murmur
<i>Non-restrictive ventricular septal defect</i> (Diameter > 1 cm/m ² BSA or orificial area > 0.8 cm ² /m ²)	RV pressure $> 75\%$ of systemic pressure
<i>Large ventricular septal defect</i>	Left to right shunt $> 2:1$

The importance of each of these features will be discussed. The size of ventricular septal defect is the most important determinant of the auscultatory findings and the clinical course of patients. Ventricular septal defects are classified as small, moderate and large not only by physical size but also by alteration in pressure and flow (Table 23.16).

**Fig. 23.20: Role of size of ventricular septal defect**

SYSTOLIC MURMURS

The murmur of the ventricular septal defect is determined to some extent by the size of the defect. In large, non-restrictive defects with equalization of pressures in left and right ventricles, the murmur may not be pansystolic, especially with pulmonary vascular disease with decreasing shunt. The loudest and longest murmur is heard in restrictive defects. A large defect with a large shunt usually results in associated diastolic mitral flow murmur. When Eisenmenger's syndrome develops in a large ventricular septal defect, the murmur disappears entirely.

Evaluation

In the evaluation of ventricular septal defect, the following questions need to be answered.

Is it VSD or a conditions simulating it?	<i>Conditions simulating VSD</i> Tricuspid regurgitation Pulmonic stenosis Mitral regurgitation Aortic stenosis
If it is VSD, what is the size?	Small, moderate, large or alternatively restrictive defect or unrestrictive defect
What is the magnitude of left to right shunt?	< 1.5:1 or more 2:1 or more
Is there pulmonary arterial hypertension? If so of what severity?	Mild Moderate Severe
In case of PAH, what type?	Hyperkinetic (flow related) Fixed (resistance related)
What is the site?	Perimembranous, inlet, subpulmonic or muscular
Is it single or multiple?	Important for surgical correction
Is it isolated VSD or is it part of a complex defect?	Tetralogy of Fallot Transposition of great arteries
Are there any associated lesions?	Aortic regurgitation Patent ductus arteriosus Pulmonary stenosis Coarctation of aorta Mitral valve disease

Timing

The murmur of ventricular septal defect is typically pansystolic and is a reflection of the pansystolic pressure difference between the two ventricles. This typical

Table 23.17: Conditions under which non-pansystolic murmurs may occur in VSD

<i>Condition</i>	<i>Mechanism</i>
Large unrestrictive VSD	Right ventricular pressures > 75% of systemic pressures
Very small VSD	Resistance at VSD itself
Muscular VSD	Closure of defect by late systole
Multiple VSDs	Summation effect as large VSD with Pulmonary hypertension
Pulmonary hypertension	Obliteration of pressure difference
Associated lesions	Elevation of right ventricular pressures
Pulmonic stenosis	
Patent ductus arteriosus	
LV inflow obstruction	
Mitral regurgitation	

murmur is a feature of small and moderate ventricular septal defects. The determinants of the timing of the murmur of ventricular septal defect are the size of the defect, the pulmonary arterial pressure, the location of the defect, and the associated defects (Table 23.18).

Site of best audibility and selective conduction

The murmur of ventricular septal defect is best audible along the left sternal border anywhere from the second to fourth spaces and is not selectively conducted anywhere (Fig. 23.21). The so-called supracristal ventricular septal defect is best heard at the pulmonary area and may be selectively conducted to the infraclavicular area and the left side of neck. The lack of right ventricle enlargement in spite of a prolonged murmur, absence of prominent *a* wave in the neck veins, and normal split and intensity of second heart sound favour a diagnosis of ventricular septal defect over pulmonic stenosis.

When the murmur of ventricular septal defect is best heard at lower left sternal border, it may simulate tricuspid regurgitation. The rough murmur, lack of any ventricular enlargement or biventricular enlargement favor a diagnosis of ventricular septal defect. A pansystolic murmur with pure right ventricle enlargement rules out ventricular septal defect and favours tricuspid regurgitation. The ventricular septal defect of L-TGA may be best audible at apex and be mistaken for mitral regurgitation. The single second heart sound and evidence of varying

SYSTOLIC MURMURS

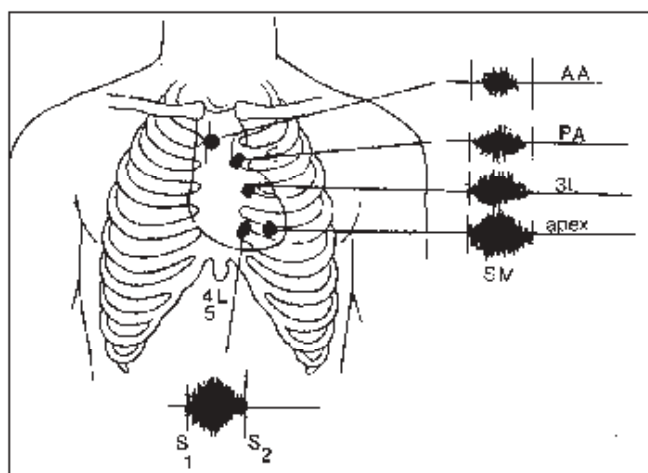


Fig. 23.21: The wide audibility of VSD murmur. Note the mid-systolic peaking unlike MR or TR

degrees of AV block (diminished first heart sound, variable first heart sound, irregular cannon waves in JVP) suggest ventricular septal defect in L-TGA. The ventricular septal defect of left ventricle to right atrium type (Gerbode's defect) may be selectively conducted to the right of the sternum or, rarely, even to the neck.

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Character and grade

The murmur of ventricular septal defect is as a rule above grade 4/6 and is almost always associated with a thrill. Thrill is uncommon with mitral regurgitation and is extremely rare with tricuspid regurgitation. Absence of thrill does not rule out ventricular septal defect. The murmur is characteristically rough or harsh in character. Rarely, it can be high-pitched or soft especially when small and muscular. This harsh character of the murmur distinguishes it from tricuspid regurgitation and mitral regurgitation, which are soft and blowing in nature.

Relation to physiologic act

The murmur is better heard during expiration and is diminished with inspiration. This behaviour of the murmur is not consistent. More importantly, the murmur fails to increase with inspiration; this distinguishes it from tricuspid regurgitation. Rarely the murmur of ventricular septal defect may appear to increase during inspiration and decrease during expiration. This unexpected behaviour is related

to the change in the orientation of the heart and interventricular septum with respiration with changes in the direction of the jet. The jet may be directed towards the site of auscultation in one phase of respiration and directed away in another. As this phenomenon is unpredictable from one patient to the other, the murmur may occasionally be better heard during inspiration. This phenomenon of directional changes in the jet ('swing') may be demonstrated during Doppler echocardiography. This 'swing' ventricular septal defect may explain some of the unusual auscultatory features of VSD mentioned above. The mitral regurgitation of mitral valve prolapse may be heard medially and may be confused for ventricular septal defect but failure of the murmur to increase on standing and Valsalva straining indicates a ventricular septal defect.

Associated features

Moderate to large ventricular septal defects with left to right shunt produce biventricular enlargement and favour the diagnosis of ventricular septal defect over tricuspid regurgitation or pulmonic stenosis. Mild to moderate pulmonary arterial hypertension can coexist with a pansystolic murmur of ventricular septal defect. In the presence of severe pulmonary arterial hypertension with right ventricle pressure, systemic or near systemic, a pansystolic murmur of ventricular septal defect is unlikely.

A pansystolic murmur in the presence of severe pulmonary arterial hypertension is more likely to be tricuspid regurgitation. The only ventricular septal defect which may retain its pansystolic timing in spite of severe pulmonary arterial hypertension is the left ventricular right atrial communication or Gerbode's defect.

The mid-diastolic murmur at the apex in ventricular septal defect, suggests that the pulmonary flow is at least twice the systemic flow.

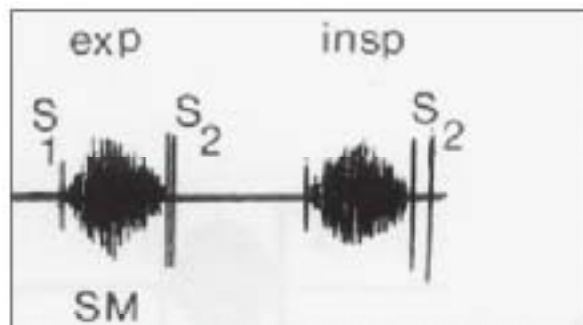


Fig. 23.22: Second heart sound split in VSD

Associated lesion

The aortic regurgitation in ventricular septal defect is usually due to a cusp prolapse (most commonly right cusp, rarely non-coronary cusp) or a bicuspid aortic valve. To cause aortic regurgitation, the defect has to be perimembranous or should have at least some degree of perimembranous extension when it is subpulmonic. The ventricular septal defect is always in the left ventricular outflow portion of the septum and is subjacent to the aortic valve. The early diastolic murmur of aortic regurgitation with ventricular septal defect gives a to and fro character to the murmur. The harsh, rough character of the systolic murmur is distinctly different from the soft, high frequency nature of the murmur of aortic regurgitation. The syndrome of ventricular septal defect with aortic regurgitation occurs in more than one condition.

- Isolated ventricular septal defect with aortic regurgitation
- Tetralogy of Fallot with aortic regurgitation
- Sinus of Valsalva aneurysm with aortic regurgitation

Mechanisms

The mechanisms by which aortic regurgitation may take place are:

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- Prolapse of aortic cusp
- Bicuspid aortic valve
- Aneurysm of sinus of Valsalva
- Congenital fenestrations of the cusp
- Infective endocarditis

It is essential to know the mechanism of aortic regurgitation in ventricular septal defect because the aortic regurgitation due to prolapse of aortic cusp is amenable to repair but a bicuspid valve requires valve replacement. The accompanying loud aortic ejection click suggests bicuspid aortic valve. Onset of aortic regurgitation in ventricular septal defect is an indication for early surgery. Even a small ventricular septal defect requires surgery once aortic regurgitation develops, as aortic regurgitation progresses over a period of time. It is for this reason, that in all patients with ventricular septal defect, one must check for an early diastolic murmur of aortic regurgitation carefully. Mild to moderate aortic regurgitation is often amenable to surgical repair but longstanding severe aortic regurgitation requires valve replacement. When the aortic regurgitation is mild,

Table 23.18: Determinants of the murmur of ventricular septal defect (VSD)

<i>Size of the defect</i>	
Small VSD	Pansystolic murmur
Moderate VSD	Pansystolic murmur
Large VSD	Early systolic or ejection systolic murmur
<i>Pulmonary arterial pressure</i>	
No pulmonary hypertension	Pansystolic murmur
Pulmonary hypertension	Early systolic or ejection systolic murmur
	No murmur
<i>Location of the defect</i>	
Muscular septum	Early systolic or ejection systolic murmur
Supracristal defect	Best heard at pulmonary area
	Selective conduction to infraclavicular area, left side of neck
	Simulates pulmonary stenosis
Left ventricular right atrial communication (Gerbode's defect)	May be conducted to the right of sternum or neck
Sub-aortic defect	May be conducted to aortic area (right 2nd space) or neck
<i>Associated defects</i>	
Pulmonic stenosis	No murmur across ventricular septal defect
PDA (small to moderate)	Continuous murmur masks the pansystolic murmur of VSD
PDA (large with PAH)	Short systolic murmur or no murmur
Coarctation (pre-ductal) with PAH	Short systolic murmur or no murmur
Coarctation (post-ductal)	Long systolic or pansystolic murmur in spite of PAH
Ruptured sinus of Valsalva into right heart	Continuous murmur masks the pansystolic murmur of VSD. The murmur may be best heard along right sternal border
L-transposition of great arteries	The systolic thrill may be absent along LSB due to side to side relation of ventricles

the features of ventricular septal defect dominate the clinical features and aortic regurgitation is often missed. When aortic regurgitation becomes severe, the ventricular septal defect is less prominent and is likely to be missed. It is good practice to look for a ventricular septal defect in all patients with aortic regurgitation either at physical examination or subsequent investigation. The systolic murmur of ventricular septal defect in the setting of severe aortic regurgitation may be mistaken for aortic stenosis, but a systolic thrill along the left sternal border, lack of carotid conduction, a collapsing pulse, and absence of features of significant aortic stenosis in spite of long systolic murmur favour the diagnosis of ventricular septal defect.

SYSTOLIC MURMURS

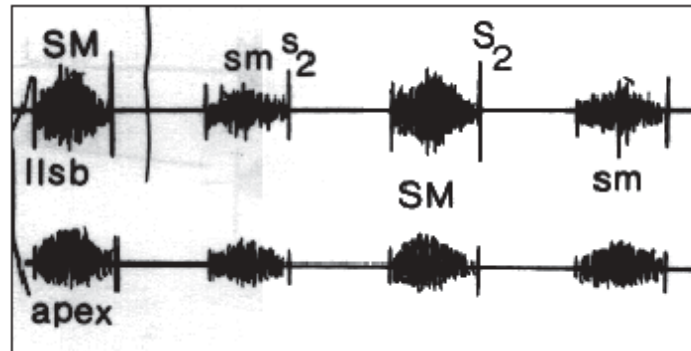


Fig. 23.23: Ventricular septal defect of acute myocardial infarction. Note alternation of murmur due to left ventricular failure.

Age of onset of murmur

The murmur of ventricular septal defect most often makes its appearance between 2 and 6 weeks after birth. A murmur appearing before 18 hours of age and later than 6 months is never due to isolated ventricular septal defect.

A new onset murmur in patients above 40 years in the setting of acute myocardial infarction suggests interventricular septal rupture causing ventricular septal defect.

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DIFFERENTIAL DIAGNOSIS OF PANSYSTOLIC MURMURS

There are only three sites in circulation where pansystolic murmurs are possible. These are mitral regurgitation, tricuspid regurgitation and ventricular septal defect. These conditions require to be differentiated from each other (Table 23.19). In actual practice, not only do these murmurs simulate each other, but other murmurs may also simulate them.

Table 23.19: Murmurs simulating pansystolic murmurs

Condition	Simulated by
Ventricular septal defect	Severe pulmonic stenosis
Mitral regurgitation	Ductus with diastolic component faint or absent
Tricuspid regurgitation	The apical systolic murmur of severe aortic stenosis Infundibular pulmonic stenosis

Table 23.20: Differential diagnosis of a pansystolic murmur

<i>Feature</i>	<i>MR</i>	<i>TR</i>	<i>VSD</i>
Site of best audibility	Apex	Tricuspid area	LSB, anywhere from pulmonary to tricuspid area
Grade	Grade 3/6 or 4/6, thrill can occur but rare	Thrill does not occur	Thrill is common
Selective conduction	Axilla and back	No selective conduction	No selective conduction
Character	Soft, blowing high frequency	Soft, blowing high frequency	Rough, harsh, combination of high and low frequencies
Relation to physiological act	Fails to increase during inspiration	Increases during inspiration	Fails to increase during inspiration
Accompanying features	LV enlargement Diminished S1 LV S3 Apical diastolic murmur	Signs of PAH RV enlargement Elevated JVP with prominent <i>v</i> wave	Biventricular enlargement Signs of PAH Apical diastolic murmur

Once a definitive pansystolic murmur is detected, one must assign it to one of the three possibilities.

In the majority of patients, it is easy to differentiate the disease from any other. Occasional difficulty arises when there is variation in the character, site or radiation of these murmurs. In rare cases, the murmur of mitral regurgitation or tricuspid regurgitation can be relatively rough simulating ventricular septal defect but the rest of the features are typical. In children, the murmur of mitral regurgitation can be best heard at the lower left sternal border and may be heard clearly at the pulmonary area simulating a ventricular septal defect. The murmur of ventricular septal defect can be of high frequency and may rarely appear to increase with inspiration as was discussed earlier.

24 Diastolic Murmurs

Unlike systolic murmurs, diastolic murmurs always signify an abnormal cardiovascular system either structurally or functionally. It is for this reason that the diastolic murmurs are generally not graded by their intensity unlike the systolic murmurs. Diastolic murmurs are often graded by their length. The presence or absence of thrill should be additionally mentioned.

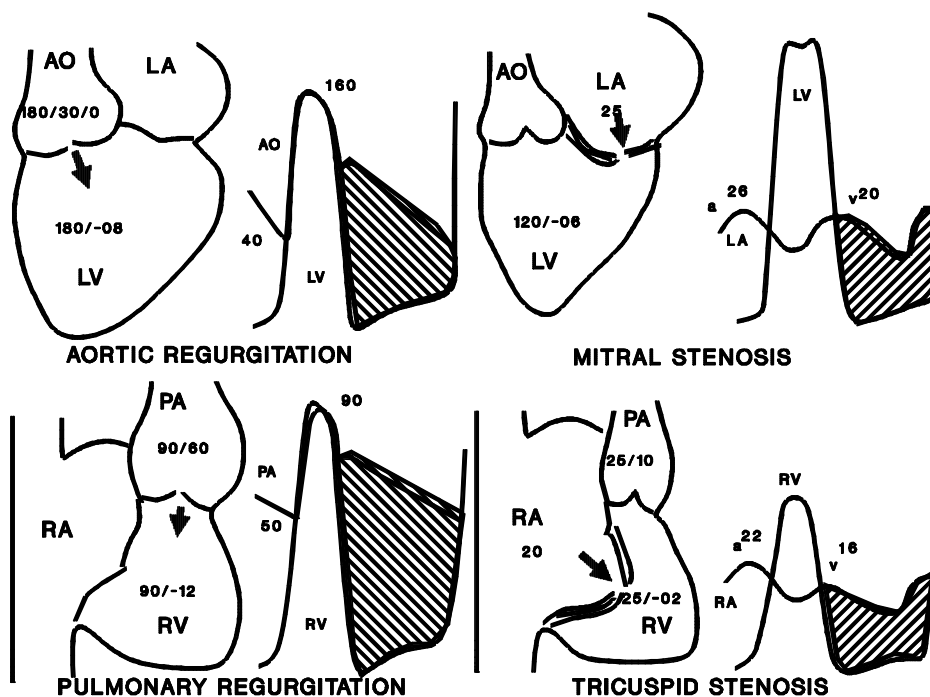


Fig. 24.1: Diastolic murmurs

Classification*Those arising at the atrioventricular valves*

- Mid-diastolic
- Pre-systolic
- Combined

Those arising at the semilunar valves

- Early diastolic
- Mid-diastolic sounding like early diastolic

These murmurs can result either at the AV valves or semilunar valves. Due to low pressure gradient at the AV valves, the murmur is of low frequency at these sites. Semi-lunar valve regurgitation on the left side is always of high frequency because of high pressure difference. However, on the right side it depends on whether pulmonary hypertension is present or not.

The murmur of pulmonary incompetence with normal pulmonary artery pressure occurs slightly later in diastole and sounds mid-diastolic.

DIASTOLIC MURMURS AT THE LEFT ATRIOVENTRICULAR VALVE

The normal flow of blood across the mitral and tricuspid valves is noiseless. Any disturbance in the normal flow pattern can result in turbulence and therefore a murmur.

Mechanisms and causes*Narrowing of mitral valve or left ventricular inflow*

- Mitral stenosis
- Left atrial myxoma
- Cor triatriatum
- Constriction of AV groove as in constrictive pericarditis
- Hypertrophic cardiomyopathy (narrow inflow cavity)

Increased flow across the AV valve

- Left to right shunts (ventricular septal defect, ductus)
- Mitral regurgitation (severe)
- Hyperkinetic circulatory states
- Chronic complete heart block

DIASTOLIC MURMURS

Mechanisms that interfere with mitral valve opening

- Austin Flint murmur with severe aortic regurgitation
- Ventricular aneurysm with a narrow neck

MITRAL STENOSIS

Mitral stenosis (MS) is almost always due to rheumatic heart disease and the stenosis results from commissural fusion and leaflet thickening. Calcification often occurs in older patients. The involvement of chordae tendinae is variable. Mitral stenosis is the commonest of valvular lesions due to rheumatic heart disease.

The murmur of MS is the most important member of this group and will be considered in detail.

Evaluation

In the evaluation of mitral stenosis the following questions need to be answered at the end of physical examination. A systematic approach to the various components of the murmur helps to answer most of the questions.

- Is it mitral stenosis or a condition simulating it?

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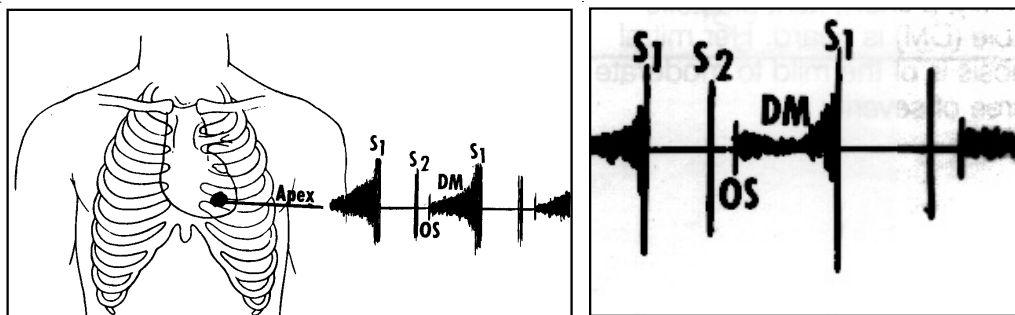


Fig. 24.2: Loud first heart sound, opening snap and mid-diastolic murmur as heard at the apex in case of mitral stenosis

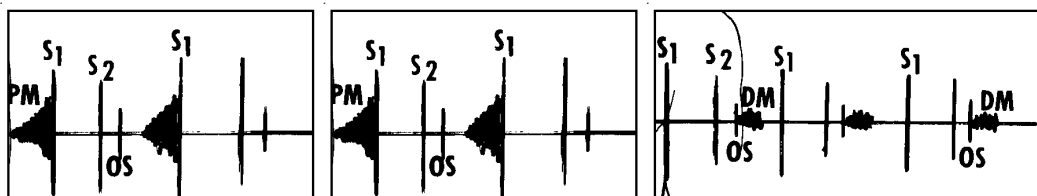


Fig. 24.3: Murmurs in mitral stenosis. Note loud S1 OS: opening snap; DM: mid-diastolic murmur following OS; PM; pre-systolic murmur that precedes loud S1

Table 24.1: Features of the murmur of mitral stenosis

<i>Feature</i>	<i>Description</i>
Site of best audibility	Apex
Timing	Mid-diastolic/pre-systolic
Selective conduction	Localised to apex
Character	Rough, rumbling (low-pitched)
Length	Short/moderate/long
<i>Relation to physiological act</i>	
Respiration	Increases during expiration
Posture	Increases in left lateral, decreases on standing
Amyl nitrate inhalation	Increases
Isotonic exercise	Increases
Isometric hand grip	Variable
<i>Accompanying features</i>	Loud first heart sound, opening snap, diastolic thrill, pulmonary hypertension, right ventricular hypertrophy, absence of left ventricular hypertrophy Dyspnea/paroxysmal nocturnal Right ventricular failure Atrial fibrillation

- If mitral stenosis, what is the severity?
- Is the valve pliable or calcified?
- Is there subvalvular fusion?
- What is the rhythm?
- Is there pulmonary hypertension? What is the severity?
- Is there right heart failure?
- Are there associated lesions?
- Is there a discrepancy between symptoms and physical signs?
- Is there a peripheral embolism?
- Is left atrial myxoma, a possibility?

As the mitral valve becomes stenotic, the left atrial pressure gets elevated with a gradient between left atrium and left ventricle in diastole (Fig. 24.4). The opening snap (OS) results from abrupt opening of the doming mitral valve. As the atrial contraction contributes to increased gradients in pre-systole, there is pre-systolic accentuation of the murmur.

DIASTOLIC MURMURS

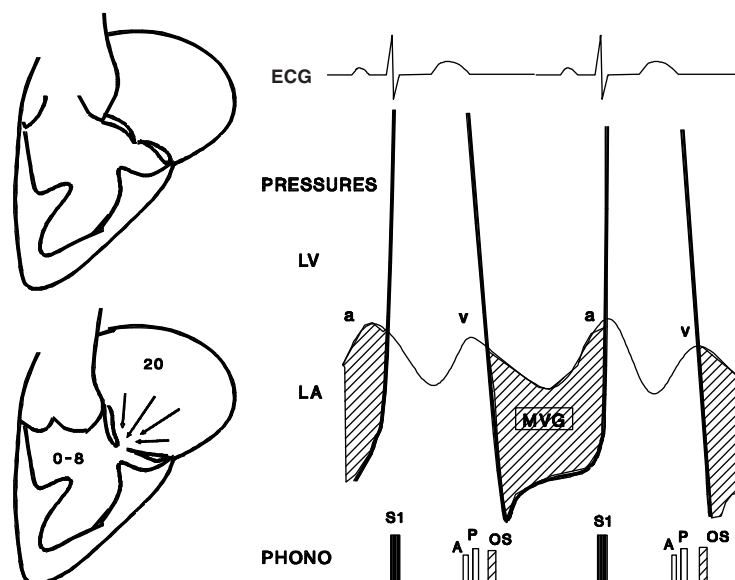


Fig. 24.4: Mechanism of mid-diastolic murmur in mitral stenosis

Timing

The mid-diastolic murmur: The murmur of mitral stenosis is mid-diastolic in timing with a pre-systolic murmur. A mid-diastolic murmur by definition means that there is a time interval between the second sound and the murmur. When the ventricle relaxes in diastole, the semilunar valves close first followed by the opening of AV valves. This time interval between semilunar valve closure and AV valve opening is called *isovolumic relaxation time* and the ventricle relaxes like a closed cavity at this time. Any murmur at the mitral or tricuspid valves in diastole can only occur after this isovolumic relaxation time. Occasionally, with severe pulmonary hypertension, the pulmonic sound may be delayed and the aortic sound may be diminished or absent due to associated aortic valve disease. In such situations, the mitral diastolic murmur may sound early diastolic. However, the character of the murmur, the site of best audibility and other features, are of help in distinguishing mitral stenosis from aortic regurgitation.

The pre-systolic murmur: Though the mid-diastolic murmur often occurs in conditions simulating mitral stenosis, the pre-systolic murmur is rare in the absence of mitral stenosis. The pre-systolic murmur of mitral stenosis may be simulated by a very loud atrial gallop that occurs with hypertrophic cardiomyopathy. In mild

mitral stenosis, a pre-systolic murmur may be the only feature in some patients. It is not known why in mild mitral stenosis, a mid-diastolic murmur occurs in some patients and a pre-systolic murmur in others. It may be related to the atrial health, the pattern of left ventricular inflow, and the nature of subvalvular apparatus.

The pre-systolic murmur is usually absent after the onset of atrial fibrillation. It must be realized that absence of pre-systolic murmur in atrial fibrillation is not a rule. In a person with very severe mitral stenosis, the left atrial pressures remain high even at the end of the diastole. In severe mitral stenosis, in spite of atrial fibrillation, a pre-systolic murmur may persist. In the short diastolic cycles the pre-systolic murmur is often heard in atrial fibrillation. However, in long diastolic cycles if the pre-systolic murmur persists, it is a reliable sign of tight mitral stenosis. Even a pre-systolic accentuation of the murmur may persist in atrial fibrillation due to progressive narrowing of the mitral valve funnel due to delayed closure of the mitral valve in mitral stenosis. The persistence of pre-systolic murmur in atrial fibrillation is not surprising, since the genesis of pre-systolic murmur is not dependent on atrial contraction alone.

Mechanisms of pre-systolic murmur

The various mechanisms are:

- Atrial contraction
- Persistent atrioventricular gradient
- Left ventricular contraction in pre-systole reducing the mitral funnel

The pre-systolic murmur or accentuation may be absent in conditions other than atrial fibrillation, like mild mitral stenosis, prolonged P-R interval, elevated LVEDP (left ventricular dysfunction) or bradycardia.

In first degree heart block with severe prolongation of P-R interval, the pre-systolic accentuation may actually occur in mid-diastole. With significant bradycardia, with long diastole, there may not be enough gradient by pre-systole. With elevated left ventricular end diastolic pressure as in left ventricular dysfunction, the pre-systolic murmur or accentuation may be absent. An atrial gallop in hypertrophic cardiomyopathy occasionally simulates the pre-systolic murmur. On the right side, the right atrial gallop in severe pulmonic stenosis can simulate a pre-systolic murmur. The length of the diastolic murmur correlates with the severity of mitral stenosis.

Table 24.3: Conditions when the length of the murmur is unreliable in mitral stenosis

<i>Condition</i>	<i>Longer/shorter</i>	<i>Mechanism</i>
Tachycardia	Longer	Diastolic abbreviation
Bradycardia	Shorter	Prolonged diastole
Low cardiac output	Shorter	Lower left atrial pressure
Severe RVF		
Severe TR		
Severe PAH		
High cardiac output	Longer	Higher left atrial pressure
Anemia		
Pregnancy		
Thyrotoxicosis		
Anxiety		
↑LVEDP	Shorter	Obliteration of transmitral gradient
Coronary artery disease		
Cardiomyopathy		
Systemic HTN		
Aortic valve disease		
Atrial fibrillation	Variable	Varying diastolic cycle lengths

Mechanisms influencing length of the murmur

- Cardiac output
- Heart rate
- Left atrial pressure
- Left ventricular end-diastolic pressure
- Heart rhythm

When there is alteration in any of the above features, the murmur of mitral stenosis should not be relied upon to assess the severity of mitral stenosis.

In the presence of any of the features in Table 24.3, the length of the murmur cannot be used to assess the severity of mitral stenosis.

Site of best audibility and conduction

The murmur of mitral stenosis is usually heard best at the apex and being low-pitched, is localized to the apex without any selective conduction. The murmur is so much localized to the apex, that if the apical impulse is missed, the diagnosis

of mitral stenosis is also missed. It is for this reason that one must go out of the way to localize the apex in the left lateral position and check for the diastolic thrill and the murmur of mitral stenosis. Occasionally, the murmur is audible only at the tricuspid area, left sternal border and even at the pulmonary area. The murmur when heard at the tricuspid area can be mistaken for the murmur of organic tricuspid stenosis. The error is compounded by the mild inspiratory increase in the murmur due to the attendant increase in heart rates during inspiration with abbreviation of diastole. Another rare but unique feature of the murmur of mitral stenosis is that it may be audible only along the left sternal border. This selective audibility only along the left sternal border may be mistaken for murmur of aortic regurgitation. However, its low frequency, the mid-diastolic timing, and lack of peripheral signs of aortic regurgitation, in spite of a long diastolic murmur, are helpful clues. The 2-D echocardiogram in these patients shows a shortened anterior leaflet and an elongated posterior leaflet composed of the tethered posterior wall of left ventricle. In effect, the diastolic jet of mitral stenosis is directed anteriorly toward the interventricular septum unlike the normal jet, which is directed towards the apex. This type of valve anatomy does not permit a closed or open commissurotomy and requires mitral valve replacement. Due to the elevated posterior leaflet by the posterior wall of left ventricle, the replaced prosthetic valve often abuts close to the left ventricular outflow. The murmur of mitral stenosis is never heard medially in the presence of right ventricular failure and dilatation.

Be that as it may, the most common murmur of mitral stenosis is localized to the apex; an occasional murmur is also heard at the tricuspid area, left sternal border or pulmonary area. Even more rarely the murmur is best heard along the left sternal border due to distorted valve anatomy, left ventricular geometry and unusually directed jet. In patients with severe emphysema, the murmur of mitral stenosis is best heard at the xiphisternal area as the cardiac impulse is available only at that site. Even the echocardiographic image can be obtained only by this view.

Character

The murmur of mitral stenosis is low-pitched, rough and rumbling in character. It sounds like a bullock cart slowly crossing a wooden bridge. The murmur is low-pitched because the pressure gradient across the valve is low even if the stenosis is

severe and the highest pressure the left atrium can attain is generally not more than 30 mmHg. A very low-frequency, loud diastolic murmur with a thrill generally indicates a pliable non-calcific mitral valve capable of low-frequency vibrations. A severely calcific immobile valve on the other hand, is not capable of low-frequency vibrations and gives rise to a higher frequency murmur with lesser intensity and no thrill. Since the murmur is low-pitched, it is better heard with the bell than with the diaphragm. This feature is of importance because the high frequency murmur of aortic regurgitation is also heard at the apex and is best heard with the diaphragm. In short, the diastolic murmur heard better with the diaphragm suggests aortic regurgitation, and the murmur heard better with the bell suggests mitral stenosis. In actual practice, this is applicable only when the bell and diaphragm are of equally good quality (equisensitive).

Relation to physiologic act or maneuvers

Respiration: The murmur of mitral stenosis is heard best during expiration in the left lateral position when the venous return to the left heart increases with the apical impulse nearer to the chest wall. However, respiratory change to the murmur is never as impressive as that of tricuspid stenosis. Occasionally, the expiratory increase is unimpressive and may actually appear to increase during inspiration. This is related to the increase in heart rate with inspiration and decrease with expiration. With the shorter diastole of inspiratory increase in heart rate, the gradient across the mitral valve increases and the murmur is exaggerated (false positive Carvallo's sign). The opposite happens in expiration.

Exercise: In mild mitral stenosis, and even in moderate mitral stenosis, with slow heart rates, the murmur may be heard only on exertion. Exercise brings in or increases the murmur by the increase in heart rate and blood flow secondary to the rise in cardiac output. Even minimal exercise such as turning the patient to the left lateral position, increases the murmur. Exercise should be used routinely when mitral stenosis is suspected. Mitral stenosis is not ruled out unless exercise is used as a maneuver. One must identify the clinical situations in which to look for mitral stenosis.

- Any patient with unexplained dyspnea
- Loud first heart sound with normal heart rate and P-R interval
- Unexplained pulmonary arterial hypertension
- All patients with rheumatic heart disease

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- All patients with aortic valve disease
- All patients with tricuspid valve disease
- All patients with atrial fibrillation
- All patients with peripheral embolism
- Unexplained left atrial enlargement/or RVH by ECG.
- All patients with 'bronchial asthma'

Amyl nitrite inhalation: Inhalation of amyl nitrite results in a fall in systemic vascular resistance and increase in heart rate. As a result, the degree of aortic regurgitation decreases, resulting in the disappearance or decrease in the Austin Flint murmur. On the other hand, the murmur of mitral stenosis increases due to increase in heart rates. In the setting of severe AR and a mid-diastolic murmur at the apex, the differential diagnosis lies between mitral stenosis and the Austin Flint murmur. Unfortunately, amyl nitrite is not available in India.

Posture: The murmur of mitral stenosis is best heard in the left lateral position and decreases on standing due to reduced venous return. The murmur of mitral valve obstruction due to left atrial myxoma on the other hand, is better heard on standing and may be lesser in intensity and duration in the supine position. This expected (or unexpected) variation in the murmur with posture is not diagnostic of left atrial myxoma and may occur with routine mitral stenosis. The accompanying tachycardia of standing is responsible for increased gradient across the valve with an increase in the murmur.

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Valsalva maneuver: The Valsalva maneuver results in decrease and disappearance of the murmur of mitral stenosis, with reappearance post release. However, the response is delayed in comparison to the murmur of tricuspid stenosis.

Accompanying features

Dyspnea/paroxysmal nocturnal dyspnea/orthopnea: The length of the diastolic murmur of mitral stenosis correlates with the severity of symptoms in mitral stenosis. In spite of mitral stenosis, if the patient has no significant dyspnea but has edema or puffiness of face as the dominant feature, associated tricuspid stenosis should be suspected. When symptoms are out of proportion to the length of the murmur, the accompanying severe pulmonary hypertension or severe right ventricular failure with tricuspid regurgitation may be responsible. A rare but important possibility is left atrial myxoma.

Loud first heart sound: The loud first heart sound calls attention to the underlying mitral stenosis because this is the easiest of physical signs to detect. In addition, this sign distinguishes mitral stenosis from other causes of mid-diastolic murmur at apex (Austin Flint murmur, the mid-diastolic murmur of pure mitral regurgitation) where the first heart sound is diminished. If the first heart sound is not loud in mitral stenosis, the following possibilities should be considered.

- Mid-diastolic murmur due to Austin Flint or severe pure mitral regurgitation
- Calcific mitral stenosis
- Severe subvalvular fusion with fibrous immobile valve
- Associated mitral regurgitation
- Associated severe aortic regurgitation
- Left atrial myxoma
- Severe right ventricular hypertrophy with masked left ventricular events
- Associated aortic stenosis, coronary artery disease or cardiomyopathy with elevated left ventricular end diastolic pressure

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A loud first heart sound generally, but not invariably, suggests a pliable, non-calcific valve. When the body of the anterior leaflet is mobile, a loud first heart sound is preserved in spite of calcification and immobility of posterior leaflet.

The opening snap and S2–OS interval: The presence of the opening snap distinguishes the mid-diastolic murmur of mitral stenosis from all the other causes of diastolic murmurs at apex. Usually, the longer the murmur of mitral stenosis, the shorter the S2–OS interval. A short murmur is accompanied by a long S2–OS. This discordant relationship between the murmur of mitral stenosis and the S2–OS is a feature of classic mitral stenosis. If the relationship is concordant (long S2–OS with long murmur and short S2–OS with short murmur), one should suspect an unusual cause for mitral obstruction, like a myxoma of left atrium.

Though mitral stenosis is the most important cause of diastolic murmur at the apex, there are many other causes for it (Table 24.4).

In the presence of severe aortic regurgitation, the apical diastolic murmur may be due to Austin Flint murmur or mitral stenosis. The accompanying features help distinguish one from the other (Table 24.5). The investigation of choice to distinguish one murmur from the other in this setting is Doppler echocardiography.

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Table 24.4: Causes and mechanisms of diastolic murmur at apex

Obstruction to left ventricular inflow	Rheumatic mitral stenosis Left atrial myxoma Congenital mitral stenosis Cor triatrium Constriction around AV groove
Increased flow across the mitral valve	Severe mitral regurgitation Left to right shunts (post-tricuspid shunts) Ventricular septal defect Patent ductus arteriosus Systemic arteriovenous fistula RSOV in to right ventricle Aortopulmonary window Aorticopulmonary fistula Truncus arteriosus Hyperkinetic circulatory states Anemia Thyrotoxicosis Pregnancy
Unusual mechanisms at the mitral valve	The jet of severe aortic regurgitation preventing opening of mitral valve (Austin Flint murmur) Mitral valvulitis of acute rheumatic carditis (Carey Coombs murmur) Narrowing of LV inflow due to severe LVH as in hypertrophic cardiomyopathy
Murmurs produced elsewhere but heard at apex	Ventricular aneurysm with a narrow neck Tricuspid stenosis Tricuspid flow murmur in atrial septal defect The murmur of aortic regurgitation
Other physical signs mistaken for mitral diastolic murmur	Third heart sound for mid-diastolic murmur Fourth heart sound for pre-systolic murmur

TRICUSPID DIASTOLIC MURMURS

A variety of conditions produce a diastolic murmur at the tricuspid valve by more than one mechanism.

Mechanisms and causes

Tricuspid stenosis is most often rheumatic and is associated with mitral stenosis. The diastolic murmur of tricuspid stenosis is similar to that of mitral stenosis.

Table 24.5: Differentiation of mitral stenosis from Austin Flint murmur

<i>Mitral stenosis</i>	<i>Austin Flint murmur</i>
Occurs with organic mitral stenosis in rheumatic heart disease	Occurs only with severe aortic regurgitation, usually non-rheumatic
Long history of dyspnea is the presenting symptom	Asymptomatic for long time or palpitation is the presenting symptom
Signs of pulmonary hypertension are common	The aortic regurgitation murmur is best heard along the left sternal border
First heart sound is accentuated	
Opening snap is diagnostic	
Third heart sound of LV origin never occurs	
Diastolic thrill is common	
Amyl nitrite increases the murmur	

However, the most important difference is the remarkable increase in intensity of the murmur with inspiration observed in tricuspid stenosis (Fig. 24.6). In fact, absence of this feature should raise doubts about the diagnosis of tricuspid stenosis.

The importance of each of these features will be discussed (Tables 24.6 and 24.7).

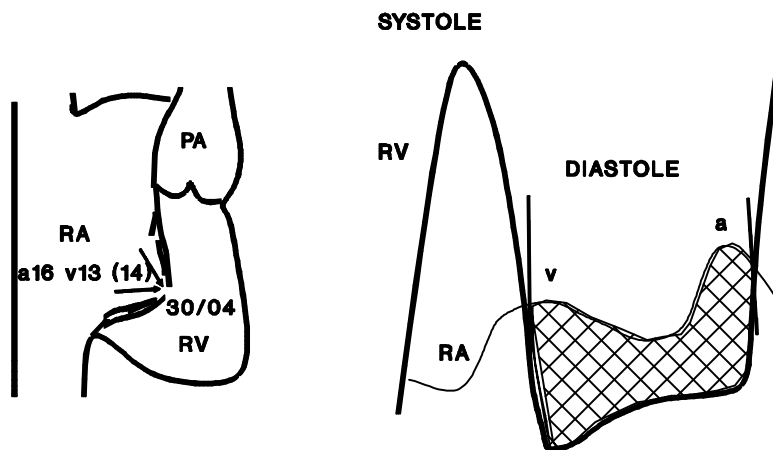


Fig. 24.6: Mechanism of diastolic murmur of tricuspid stenosis

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Table 24.6: Mechanisms and causes of tricuspid diastolic murmurs

<i>Mechanisms</i>	<i>Causes</i>
Obstruction to right ventricular inflow	Tricuspid valve stenosis Rheumatic Congenital Carcinoid Right atrial tumours Myxoma Secondary tumours Ebstein's anomaly
Increased flow across the valve	Pre-tricuspid shunts Atrial septal defect TAPVC RSOV into RA LV to RA communication Coronary artery to RA communication Lutembacher's syndrome Partial anomalous venous connection
Interference with opening of tricuspid valve	Severe tricuspid regurgitation Functional Organic
Murmurs produced elsewhere but also heard at tricuspid area	Severe pulmonary regurgitation with right sided Austin Flint murmur Mitral stenosis Pulmonary regurgitation Aortic regurgitation
Murmurs mistaken for tricuspid diastolic murmur	Normal pressure pulmonary incompetence Pericardial rub Right sided S4 may sound like pre-systolic murmur

Table 24.7: Features of the murmur of tricuspid stenosis

<i>Feature</i>	<i>Description</i>
Site of best audibility	Tricuspid area
Timing	Mid-diastolic
Length	Short/moderate/long
Character	Rough/rumbling
Selective conduction	Localised to tricuspid area
<i>Relation to physiological act</i>	
Respiration	Increases during inspiration
Posture	Increases in supine, passive leg raising
Rapid deep breathing	Increases

Site of best audibility

The murmur of tricuspid stenosis is usually best audible at the tricuspid area (4th space, left sternal border), but is also occasionally heard at the third space, and therefore can be mistaken for the early diastolic murmur of aortic regurgitation or pericardial rub. The murmur may occasionally be best heard at the apex in rheumatic tricuspid stenosis when the right atrium is grossly dilated displacing the tricuspid valve toward the apex. In Ebstein's anomaly, the displaced tricuspid valve (towards the right ventricular apex) and the dilated right atrium together are responsible for the tricuspid murmurs being best heard at apex.

Timing

The commonest murmur of tricuspid stenosis is pre-systolic with or without the mid-diastolic component. The length of the murmur is directly related to the severity of tricuspid stenosis. In general, the tricuspid diastolic murmurs are earlier in diastole than their counterparts at the mitral valve. For this reason, the tricuspid diastolic murmurs are often mistaken for the early diastolic murmurs of aortic or pulmonary incompetence. However, their dramatic increase with inspiration and decrease or disappearance with standing or expiration, distinguish tricuspid stenosis from aortic regurgitation and pulmonic regurgitation.

Though the length of the murmur is directly related to the severity of tricuspid stenosis, in Ebstein's anomaly due to associated atrial septal defect and in rheumatic heart disease due to associated pulmonary hypertension and elevated right ventricular end diastolic pressure, the murmur may be shorter for the degree of tricuspid stenosis.

Causes of short or no murmur

The causes of a short murmur or no murmur could be:

- Rheumatic tricuspid stenosis with accompanying mitral stenosis, severe pulmonary hypertension, elevated right ventricular end diastolic pressure
- Diuretic therapy in tricuspid stenosis
- Atrial fibrillation (absent pre-systolic murmur)
- Ebstein's anomaly of tricuspid valve

Character

The murmur of tricuspid stenosis is rough and rumbling and of low frequency. This is due to low pressure difference between the right atrium and the right

ventricle in diastole. The murmur is often mistaken for a pericardial rub due to this rough character. However in severe tricuspid stenosis particularly in right atrial tumours, the murmur assumes a higher frequency to resemble the murmur of aortic regurgitation.

Case summary

A 50-year-old man is seen first in 1979 for a heart murmur and a diagnosis of cardiomyopathy was made. On repeated follow up in various institutions including the author's, he was labeled to have pulmonary incompetence or aortic incompetence as the murmur was interpreted as high frequency or soft. An echocardiogram was not done as the facility was not available then. In 1984 the patient was evaluated by echocardiogram with Doppler and was found to have a large right atrial myxoma, which was surgically removed. Even at this time the murmur was interpreted as pandiastolic and was higher in frequency for a murmur of tricuspid stenosis.

Relation to a physiologic act

The murmur of tricuspid stenosis always increases during inspiration. The inspiratory increase is often so explosive that it is mistaken for a pericardial rub. In some patients it may be heard only during inspiration. If ordinary inspiration fails to bring in the murmur, passive leg raising in supine position during inspiration generally brings in the murmur. Another maneuver is to ask the patient to breathe in and out rapidly 5–6 times and then listening for the murmur during inspiration. The few rapid breaths increase the venous return which accumulates in the right atrium enhancing the gradient and thereby the murmur. The inspiratory increase is so consistent in tricuspid stenosis that if this feature is absent the diagnosis of tricuspid stenosis is highly unlikely.

Accompanying features

The accompanying features are important as they are often the pointers to the physician to look for the murmur of tricuspid stenosis.

The features in Table 24.8 are expected in classic tricuspid stenosis, but exceptions are common. Significant pulmonary hypertension can occur in spite of tricuspid stenosis. The *a* wave in the jugular venous pulse is absent in atrial fibrillation and the slow *y* descent is an unreliable sign. Many patients with milder degrees of tricuspid stenosis do not present with facial or leg swelling, particularly, if they are receiving diuretics. Paroxysmal nocturnal dyspnea though rare in the

Table 24.8: Associated features of murmur of tricuspid stenosis

<i>Negative</i>	<i>Positive</i>
Absence of paroxysmal nocturnal dyspnea	Puffiness of face/edema of legs
Significant dyspnea	Prominent <i>a</i> and slow <i>y</i> in JVP
Absence of right ventricular hypertrophy	Tricuspid opening snap?
No pulmonary hypertension	
No right ventricular third or fourth sounds	Associated mitral stenosis

presence of tricuspid stenosis, can occur if the accompanying mitral stenosis is tight or the tricuspid stenosis is mild.

OTHER MID-DIASTOLIC MURMURS AT THE AV VALVES

MITRAL REGURGITATION

This murmur occurs even in absence of mitral stenosis when the mitral regurgitation is severe. The associated third heart sound rules out mitral stenosis. The pre-systolic component as a rule is absent. A diastolic thrill is rare but can occur in rheumatic mitral regurgitation even if mitral stenosis is absent. In fact this mid-diastolic murmur in mitral regurgitation (MR) is most common with rheumatic mitral regurgitation and is rare with non-rheumatic causes of mitral regurgitation. If the mid-diastolic murmur of mitral regurgitation is unaccompanied by S3, or is accompanied by an opening snap, associated mitral stenosis is likely. Other features are:

- Mid-diastolic and short murmur
- Never pre-systolic
- Suggests severe mitral regurgitation
- Favours rheumatic mitral regurgitation
- May have a diastolic thrill without mitral stenosis but favours rheumatic MR
- Accompanied by third heart sound
- First sound is usually diminished or absent

Severe mitral regurgitation without mid-diastolic murmur is usually non-rheumatic. When there is associated atrial septal defect, the mid-diastolic murmur at the mitral valve may not occur in spite of severe mitral regurgitation. In rheumatic mitral

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valve disease, some degree of anatomical narrowing of the valve is common though it is physiologically unimportant. The valve area may be more than 2.5 cm² but less than the normal 4 cm². When flow across this valve is increased as in severe mitral regurgitation, a flow murmur is easily produced.

The belief that in this setting a diastolic thrill always means mitral stenosis is not valid.

LEFT TO RIGHT SHUNTS

At the tricuspid valve: In the absence of organic tricuspid valve disease, a diastolic murmur at the tricuspid valve suggests increased flow across the tricuspid valve, which is twice the normal. The tricuspid diastolic murmur is best heard at the lower left sternal border. Sometimes it can be heard at the apex when the right ventricle forms the apex and the murmur is confused for mitral stenosis.

The tricuspid flow murmur in atrial septal defect is: Best heard at the lower left sternal border but may be heard at the apex or upper left sternal border; only mid-diastolic murmur is present, with no pre-systolic murmur; relatively soft or medium frequency (not rough as in tricuspid stenosis); no significant change with respiration; indicates pulmonary flow to be twice the systemic flow or higher; more specific sign of atrial septal defect than other signs; always present in Lutembacher syndrome; can occur in any pre-tricuspid shunt.

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Occasionally the murmur is audible slightly higher up along the left sternal border (third space) and is confused for aortic or pulmonary regurgitation. This error is enhanced due to the relatively softer character and higher frequency of this murmur in comparison to the murmur of tricuspid stenosis. It should be realised however, that this tricuspid diastolic murmur though is more specific than any other sign of atrial septal defect, is by no means pathognomonic of this defect. It can occur in any pre-tricuspid shunt (left to right shunt located proximal to tricuspid valve).

Causes

- Left to right shunts (pre-tricuspid)
 - Atrial septal defect (secundum/primum defects)
 - Partial anomalous venous connection
 - Rupture of sinus of Valsalva in to right atrium
 - Coronary cameral fistula into right atrium

- Left ventricular right atrial communication (Gerbode's defect)
- Admixture lesions (cyanotic heart disease)
 - Total anomalous pulmonary venous connection
 - Single atrium
 - Hypoplastic left heart syndrome (mitral atresia)
- Severe tricuspid regurgitation
- The right sided Austin Flint murmur in severe functional pulmonary regurgitation

Unlike the murmur of tricuspid stenosis, the flow murmur in these settings is usually only mid-diastolic, softer and does not increase significantly during inspiration.

Increased flow across the mitral valve

When the flow across the normal mitral valve is twice the normal flow, a mid-diastolic murmur occurs in a variety of states.

Left to right shunts (post-tricuspid shunts)

- Ventricular septal defect
- Patent ductus arteriosus
- Aortopulmonary window
- Systemic arteriovenous fistula
- Admixture lesions (cyanotic heart disease)

Increased pulmonary flow

- Double outlet right ventricle
- Single ventricle
- Truncus arteriosus
- Tricuspid atresia with large ventricular septal defect but no pulmonic stenosis
- Extensive bronchopulmonary collaterals in pulmonary atresia or any cyanotic heart disease with diminished pulmonary flow
- Systemic to pulmonary artery shunts

Diminished pulmonary flow

- Tricuspid atresia with pulmonic stenosis

Hyperkinetic circulatory states

- Severe anemia
- Thyrotoxicosis

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Severe mitral regurgitation

In a left to right shunt such as ventricular septal defect or PDA, this mid-diastolic murmur indicates that pulmonary flow is at least twice the systemic flow and is a definite indication for hemodynamic evaluation and surgery. This physical sign also distinguishes ventricular septal defect from pulmonic stenosis and other conditions simulating it. This murmur needs to be distinguished from the third heart sound often heard in children.

AUSTIN FLINT MURMUR

This murmur can be mid-diastolic/pre-systolic and occurs in the presence of moderate to severe aortic regurgitation. It is due to the heavy jet of aortic regurgitation impinging on the anterior leaflet of mitral valve preventing adequate opening of the valve and creating turbulence at the mitral valve in diastole. Austin Flint in 1862 described this mitral diastolic murmur in patients with aortic regurgitation. He ascribed this murmur to flow from left atrium to ventricle. In 1972 Fortuin and Craige described echo-phonocardiographic studies in 15 patients with moderate to severe pure aortic regurgitation. The murmur occurs while the mitral valve is closing, and terminates at the time of first heart sound. The rapidity of the closing slope of the valve in diastole is increased by the dual influx from the atrium and aorta, thus creating a baffle to antegrade flow (Craige). With isometric hand grip, the degree of aortic regurgitation increases due to elevated peripheral vascular resistance and the Flint murmur increases. With amyl nitrite inhalation or administration of vasodilators, the murmur decreases or disappears due to the reduction in severity of aortic regurgitation. The mitral valve has to be open for this murmur to occur. In extremely severe aortic regurgitation with premature closure of the mitral valve in late diastole, the murmur is confined to mid diastole and the pre-systolic murmur does not occur. Another reason may be that all the flow from the left atrium must occur in mid-diastole thereby increasing the turbulence to flow.

Other features are:

- Can be mid-diastolic and/or pre-systolic
- Does not occur with mild aortic regurgitation
- Requires at least moderate aortic regurgitation

Table 24.9: Differentiation of Austin Flint murmur from the murmur of mitral stenosis

<i>Feature</i>	<i>Austin Flint</i>	<i>Mitral stenosis</i>
Diastolic thrill	Rare	Common
Amyl nitrite inhalation	Decreases/disappears	Increases
Isometric hand grip	Increases	Variable
Vasopressors	Increases	Variable
First heart sound	Decreased/normal	Increased
Opening snap	Absent	Present
LV third heart sound	May occur	Never occurs
Rhythm	Sinus rhythm	Atrial fibrillation is common
Diastolic pressure < 40 mmHg with pre-systolic murmur	Unlikely	Likely
Significant pulmonary hypertension	Unlikely	Likely

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- Due to the heavy jet of aortic regurgitation falling on AML preventing adequate opening and turbulence to flow across mitral valve in diastole
- With premature closure of mitral valve as in free severe AR or acute aortic regurgitation, the pre-systolic murmur does not occur
- Increases with isometric hand grip or vasopressors
- Decreases with amyl nitrite inhalation or vasodilators
- Low-pitched, heard best with the bell, unlike the murmur of aortic regurgitation which is high-pitched and heard best with the diaphragm

Disappearance of the preexisting pre-systolic Flint's murmur in an aortic regurgitation may indicate aggravation of aortic regurgitation or onset of left ventricular failure with elevated ventricular end-diastolic pressures. The Flint's murmur needs to be carefully distinguished from that of mitral stenosis (Table 24.9). In actual practice check for more than one distinguishing feature. In some patients, it is not possible to differentiate one from the other by clinical features alone. Echocardiography is diagnostic in this setting.

OTHER PHYSICAL SIGNS SIMULATING MID-DIASTOLIC MURMURS

Other auscultatory signs, occurring in the diastole can mimic the mid-diastolic and the pre-systolic murmurs at the AV valves.

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- Third heart sound as mid-diastolic murmur
- Fourth heart sound as pre-systolic murmur
- Third heart sound and fourth heart sound together as mid-diastolic murmur
- Pericardial knock of constrictive pericarditis
- Pericardial rub
- Early diastolic murmur of aortic regurgitation at apex

In hypertrophic cardiomyopathy, the loud fourth heart sound can mimic the pre-systolic murmur. More commonly, the physiological third heart sound of children is mistaken for mid-diastolic murmur. Rarely, the pre-systolic gallop (fourth heart sound) in severe pulmonic stenosis can resemble the pre-systolic murmur of tricuspid valve.

SEMILUNAR VALVE REGURGITATION

Normally, the second heart sound (A2 and P2) marks the closure of the semilunar valves on either side of the heart. Also, there is no back flow. When these valves are incompetent, the murmurs start flush with the corresponding second heart sound. It is for this reason these murmurs are called early diastolic murmurs in contrast to those arising at the AV valves which occur some time later in diastole (mid-diastolic).

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AORTIC REGURGITATION

The competence of the normal aortic valve is maintained by a combined mechanism of the aortic valve apparatus.

- Aortic valve cusps
- Commissures
- Aortic valve annulus
- Aortic root/sinuses of Valsalva
- LV contractility

Any alteration in the above mechanisms may produce aortic valve incompetence. Rheumatic heart disease is the commonest cause of aortic regurgitation it affects the valve cusps. Rarer causes like syphilis produce aortic regurgitation by dilatation and distortion of the aortic root (Table 24.10).

Table 24.10: Mechanisms of aortic regurgitation

<i>Abnormality of cusp/commissures</i>	
Reduction in area	Rheumatic fever Rheumatoid disease Ankylosing spondylitis
Perforation	Infective endocarditis
Commissural fusion	Congenital Rheumatic
Loss of commissural support	Congenital Tetralogy with AR VSD with AR Dissection of aorta
Fixed orifice	Calcific AS
Distortion of cusps	Subvalvular fixed AS
<i>Abnormality of aortic root</i>	
Aortic root distortion	Nonspecific aortitis Ankylosing spondylitis Syphilis Non specific urethritis (Behcet's) Rheumatoid disease Aortic dissection
Aortic root dilatation	All disorders with aortitis Aortopathy (non-inflammatory) Marfan's Familial Idiopathic
Intimal flap prolapse	Ehlers–Danlos Pseudoxanthoma elasticum
Functional	Aortic dissection Severe systemic hypertension

Evaluation

The questions to be answered when evaluating aortic regurgitation are:

- Is it aortic regurgitation or a condition simulating it?
- If aortic regurgitation, is it due to aortic root disease or valve disease?

Timing

- What is the severity?
- Are there associated lesions?
- Is there heart failure?

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Table 24.11: Features of the murmur of aortic regurgitation

<i>Features</i>	<i>Description</i>
Timing	Early diastolic
Site of best audibility	Right 2 nd space/LSB/apex/right sternal border
Character	High frequency/soft/blowing/musical
Length/grading	< Grade 3/6, thrill is rare
<i>Relation to physiological act</i>	
Respiration	Best heard in sitting (or standing) leaning forward,
Posture	Held expiration
Isometric hand grip	Increases
Vasopressor (phenylephrine)	Increases
Amyl nitrite inhalation or sublingual nitrate	Decreases
Prompt squatting	Increases
<i>Associated features</i>	
Fever/clubbing/recent aggravation of symptoms	Infective endocarditis
Recent/sudden aggravation in severity of AR or symptoms	Retroversion or prolapse of aortic cusp/ aortic dissection /hypertension
Arterial pulse	High volume/collapsing/bisferiens
Blood pressure	High pulse pressure/low diastolic pressure/ positive Hill's sign
LV enlargement	Hyperkinetic LV impulse
First sound	Normal with mild AR/diminished or absent in severe AR
Second sound	Normal split with mild AR/reversed split with severe AR or LV dysfunction. A2 is diminished or absent in valve disease as in rheumatic. Accentuated with aortic root disease as in syphilis
Third heart sound	Suggests heart failure or associated mitral regurgitation
Fourth heart sound	Acute AR/aortic dissection with past hypertension and left ventricular hypertrophy
Ejection systolic murmur	Occurs with moderate to severe AR commonly
Pulmonary arterial hypertension	Rare

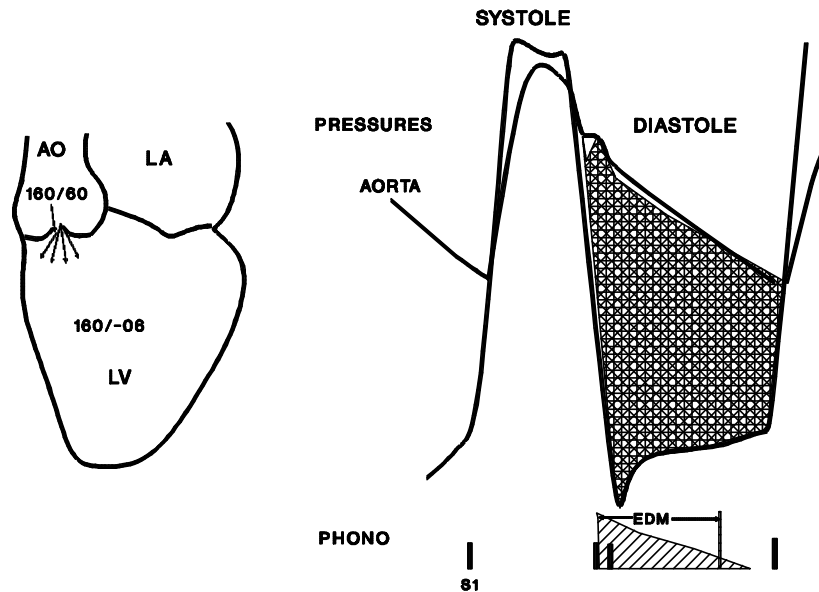


Fig. 24.7: Early diagnostic murmur in aortic regurgitation

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- Is there systemic hypertension?
- Is it chronic/acute or acute or chronic?
- Is there infective endocarditis?
- Is the patient symptomatic?

Many of these questions can be answered by the time one finishes auscultation in aortic regurgitation, if the auscultation is done systematically.

The murmur of aortic regurgitation is early diastolic starting flush with the aortic component of the second heart sound (Fig. 24.7). The early diastolic murmur of pulmonary incompetence starts later with the pulmonary components of second heart sound. This feature is hardly helpful in differentiating these murmurs from one another as the two components of second heart sound are difficult to detect in this setting. With severe pulmonary hypertension due to pulmonary incompetence, the second heart sound is often single; in aortic regurgitation, the P2 is covered by the murmur. When transmitted to the apex, the murmur is mistaken for mitral diastolic murmur. As happens often, when it is difficult to be sure if the murmur is early diastolic or mid-diastolic, the bell and the diaphragm can be used to advantage. A diastolic murmur, heard best with the bell is the mid-diastolic

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Table 24.12: Causes of murmurs at left and right sternal borders in aortic regurgitation

<i>Left sternal border murmurs</i>	<i>Right sternal border murmurs</i>
Rheumatic heart disease	Syphilis
Congenital bicuspid valve	Marfan syndrome
Infective endocarditis	Annuloectasia of aortic root
AR in association with valvular AS	Ankylosing spondylitis
AR in association with subvalvular fixed AS	Rheumatoid arthritis
	Reiter syndrome
Prosthetic AR	AR in association with tetralogy of Fallot
	AR in association with VSD

murmur of mitral valve, and the one heard best with the diaphragm, is the early diastolic murmur of aortic regurgitation.

Site of best audibility

The commonest form of aortic regurgitation (rheumatic) due to aortic valve disease, is heard best along the left sternal border but is also heard well at the right second space and apex (Table 24.12). In unusual and rare causes of aortic regurgitation (syphilitic) due to aortic root disease, the murmur is heard best along the right sternal border but is also heard along the left sternal border and apex. The audibility of the early diastolic murmur at the right second space and apex distinguishes aortic regurgitation from pulmonary incompetence.

The left sternal border murmur is suggestive of valvular origin for the murmur. With a dilated aortic root, the murmur is conducted more to the right sternal border than to the left. In the aortic regurgitation of tetralogy due to the dextroposed aortic root, the jet of aortic regurgitation may be directed selectively into the right ventricle and may result in a murmur better audible along the right sternal border. A similar phenomenon may occur in aortic regurgitation associated with ventricular septal defect, when the right coronary cusp prolapses into the right ventricle due to lack of support.

Character and diastolic thrill

The early diastolic murmur of aortic regurgitation is high-frequency, soft, blowing and may be musical in character. This is related to the high pressure difference between the aorta and the left ventricle in diastole. This is unlike that of the mid-diastolic murmur of mitral stenosis where the diastolic pressure difference is

lesser. The early diastolic murmur may assume a musical quality when the cause is infective endocarditis with vegetations over the aortic valve, or when the aortic cusp is retroverted as in syphilis. Very rarely, the murmur may be rough in character like that of aortic stenosis and may have a thrill. This almost always suggests a retroverted aortic cusp as in syphilis. This unexpected thrill of aortic regurgitation is often mistaken for a systolic thrill and some other diagnosis may be considered. However, careful auscultation with simultaneous palpation of the carotid impulse or 'inching' the stethoscope from the base to the apex is helpful.

Length of early diastolic murmur

As a general rule, the length of the murmur correlates with the severity of aortic regurgitation. However, there are many exceptions to this rule. The length of the murmur is a reflection of the duration of the pressure gradient between the aorta and the left ventricle in diastole. In the absence of heart failure in chronic aortic regurgitation, a short murmur generally indicates mild aortic regurgitation and a longer murmur is associated with moderate to severe aortic regurgitation. The length of the early diastolic murmur in aortic regurgitation is a function of aortic pressure, the degree of abnormality at the aortic valve, the left ventricular end diastolic pressure and probably the direction of the jet of aortic regurgitation. The length of the murmur is influenced by:

- Aortic pressure (systemic vascular resistance)
- Left ventricular diastolic pressure
- The actual defect at the aortic valve
- The severity of aortic regurgitation

For a given degree of defect at the aortic valve, the longer the murmur the more severe the aortic regurgitation. Again, for a given defect at the aortic valve, the higher the aortic pressure, the longer the murmur. The higher the left ventricular pressure, the shorter the murmur.

Causes of significant AR with short or no murmur are:

- Acute aortic regurgitation
- Left ventricular failure
- Tachycardia
- Hypotension
- Vasodilators
- Pregnancy

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Table 24.13: Causes and clinical settings for acute aortic regurgitation

<i>Cause(s)</i>	<i>Clinical setting</i>
Infective endocarditis	Fever Acute pulmonary edema, heart failure Rheumatic heart disease, Bicuspid aortic valve
Surgical or balloon dilatation	Deterioration after procedure
Blunt injury chest	
Trauma	
Acute aortic dissection	Acute chest pain/back pain Absent or asymmetry of peripheral pulses
PTCA: Guiding catheter interfering with aortic valve closure mechanism (especially Amplatz catheter)	Unexplained hypotension or elevation of PA diastolic pressures during PTCA

In acute severe aortic regurgitation, the left ventricular diastolic pressure inevitably rises and approximates that of aorta, abbreviating or abolishing the murmur. Even a short murmur should be given importance in the clinical setting when acute aortic regurgitation is likely.

During PTCA, the stiff guiding catheter abutting over the aortic valve may interfere with its closure and may result in significant acute aortic regurgitation. This often, manifests not as an auscultatory sign but as an unexplained hypotension with rise in pulmonary artery diastolic and systolic pressures. Initially, the aortic diastolic pressure falls; later the systolic pressure falls (Table 24.13).

Tachycardia, of both sinus and ectopic origin, shortens the murmur due to abbreviation of diastole. With hypotension or vasodilators the driving pressure for aortic regurgitation is diminished with a decrease in the murmur. In pregnancy due to hormonally induced reduction in systemic vascular resistance and the placenta acting like an arterial venous fistula, the murmur may shorten or disappear.

Relation to a physiologic act

The murmur of aortic regurgitation can be very difficult to hear and requires to be checked through certain maneuvers. Some of these maneuvers help recognise the murmur when previously audible, and others to differentiate it from murmurs that simulate it. The early diastolic murmur of aortic regurgitation is most difficult to hear when it is acute or very mild. Certain disorders are often associated with

Table 24.14: Conditions in which one must check for aortic regurgitation

<i>Condition</i>	<i>Significance</i>
Aortic stenosis	Localises the lesion to the valve or subvalvular region Rules out subvalvular dynamic obstruction (HOCM) Helps to distinguish the murmur of AS from PS Contraindicates balloon dilatation
Mitral valve disease	Favours rheumatic heart disease
Ventricular septal defect	Early surgery prevents progression of AR Indication for surgery even with a small VSD When a murmur of AR is heard the VSD murmur is mistaken for AS
Tetralogy of Fallot	Can precipitate CCF Repair or replacement of valve
Coarctation of aorta	Associated bicuspid valve Worsening of clinical course Early surgery for coarctation
Bicuspid aortic valve	Associated AR increases the risk of infective endocarditis Appearance of new murmur when the patient comes back with fever
Patient with acute chest pain	Aortic dissection is likely
Patient with fever	Infective endocarditis
Acute pulmonary edema of unknown origin	Acute AR/surgery is life saving
Coronary artery disease	Rule out aortic dissection Contraindication to intra-aortic balloon
High volume pulse or hyperkinetic circulatory state	AR is an important cause
Systemic hypertension	AR runs a rapidly progressive course Vasodilators/ACE inhibitors are the choice Can be functional with a diastolic pressure above 120 mmHg

aortic regurgitation; one must check for these (Table 24.14). In some clinical situations, detection of the murmur has important diagnostic and immediate therapeutic significance.

In all the above situations one must make the patient sit, lean forward and

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Table 24.15: Maneuvers used to bring out the early diastolic murmur of AR

<i>Maneuver</i>	<i>Mechanism</i>
Sitting, leaning forward, held expiration, diaphragm firmly applied to the chest	Aorta nearer to chest Non-interference with the noise of breathing Improved quality of diaphragm to appreciate the high-frequency murmur
Prone position	Aorta nearer to chest
Prompt squatting	Increased systemic vascular resistance
Isometric hand grip	As above
Vasopressors (phenylephrine)	Increased systemic resistance The reflex bradycardia with phenylephrine is best suited for auscultation of aortic regurgitation murmur

during held expiration with the diaphragm firmly applied to the chest, auscultate for the early diastolic murmur of aortic regurgitation when it was not audible earlier. The case of a patient with fever as a presenting manifestation deserves particular attention. It is often taught that in a patient with fever, one must look for evidence of infective endocarditis when the fever is of a few weeks duration. It must be realised that infective endocarditis has its beginnings, like any other cause, in fever. It is now well known that when the diagnosis and therapy are delayed beyond the first few days or weeks, peripheral embolism and heart failure are more likely and the chances of medical therapy succeeding are less and less likely. *One must make it a habit to look for the murmur in the heart whether the fever is one day old or many weeks old.* It is in this setting of a febrile patient with tachycardia where a murmur is not initially heard that one of the maneuvers listed in Table 24.15 must be used.

Once the murmur of aortic regurgitation is detected, the next step is to differentiate it from the other murmurs or auscultatory events which closely simulate it (Table 24.16).

The murmur of aortic regurgitation is one of the most difficult to detect and requires careful auscultation and use of various maneuvers.

Accompanying features

A high volume or collapsing pulse not only supports but also helps quantify aortic regurgitation. The absence of a collapsing pulse does not rule out aortic regurgitation

Table 24.16: Auscultatory events or murmurs simulating AR

<i>Auscultatory event/murmur</i>	<i>Differentiating feature</i>
Pulmonary regurgitation with pulmonary hypertension (Graham Steell murmur)	Not audible at right side of sternum and apex May increase with inspiration Decreases with standing/expiration Associated with severe pulmonary hypertension No peripheral signs of aortic regurgitation In the setting of valvular disease, occurs only with severe mitral stenosis
Mid-diastolic murmur of severe mitral stenosis at apex and occasionally along LSB	Low frequency, better heard with bell
Mid-diastolic murmur of severe mitral regurgitation when heard along left sternal border	As above
Mid-diastolic murmur of tricuspid stenosis	Decreases or disappears with sitting, standing, during expiration Increases with supine position, inspiration Better heard with bell Prominent <i>a</i> wave with elevated JVP
Pericardial friction rub when high frequency or musical	Changes with posture/respiration Never heard to the right of sternum Primary cause for pericarditis is generally evident

as in mild aortic regurgitation. Similarly, a collapsing pulse does not rule out pulmonary regurgitation in the presence of significant mitral stenosis and severe pulmonary arterial hypertension. Ejection systolic murmur of grade 3/6 to 4/6 is common with moderate to severe aortic regurgitation even in the absence of aortic stenosis. If the diastolic pressure is less than 40 mmHg, aortic stenosis of any significance is unlikely even if the ejection systolic murmur is grade 4/6 (Fig. 24.8).

This murmur may have a thrill over the carotid but absence of thrill over the aortic area or left sternal border and a forcible non-sustained apical impulse with a diastolic blood pressure of 40 mmHg or less argues against aortic stenosis.

Aortic vascular click is common with aortic regurgitation due to aortic root disease. This click is usually not very loud, and the accompanying loud A2 and the early diastolic murmur to the right of the sternum favour a vascular click. A loud valvular ejection click is common in aortic regurgitation due to bicuspid aortic valve or the quadricuspid valve of truncal regurgitation in truncus arteriosus. In

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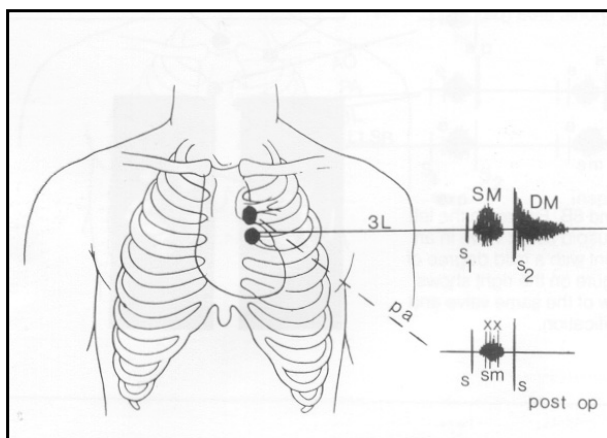


Fig. 24.8: Grade 4/6 ejection systolic murmur in AR

the absence of impressive cyanosis and clubbing, the last condition may be mistaken for pure aortic regurgitation or rheumatic heart disease in a grown up child or adolescent.

PULMONARY REGURGITATION

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Unlike aortic regurgitation, pulmonary regurgitation (PR) occurs in two settings, both of which have an important influence on the clinical features of the murmur.

- Pulmonary regurgitation with pulmonary arterial hypertension
- Pulmonary regurgitation with normal pulmonary arterial pressures

The most common form is related to high pulmonary arterial pressures and will be described first. This murmur is also called the Graham Steell murmur.

Unlike the aortic annulus, which is capable of tolerating very high pressures, the pulmonary annulus has a weak base and cannot withstand very high pressures. This murmur generally means that the pulmonary arterial pressures are equal to or higher than the systemic pressures. Each of the features listed above will be described.

Timing

The murmur of functional pulmonary incompetence is early diastolic in timing and needs to be distinguished from aortic regurgitation (Table 24.17). Theoretically, the murmur of aortic regurgitation starts flush with the aortic component of the

Table 24.17: Murmur of pulmonary incompetence in pulmonary hypertension

<i>Feature</i>	<i>Description</i>
Timing	Early diastolic
Length	Very short to pandsystolic
Site of best audibility	Pulmonary area (left 2 nd space or 3 rd space)
Character	High pitched
Conduction	Left sternal border 3 rd and 4 th spaces Apex if formed by right ventricle
<i>Relation to physiological act/maneuvers</i>	
Respiration	May increase during inspiration but often fails to do so; better heard in supine posture, passive leg raising
Posture	Diminishes or disappears in standing posture
Isometric hand grip/vasopressor	No influence
Amyl nitrite inhalation	No influence/may decrease
<i>Associated features</i>	
Signs of severe pulmonary hypertension	Loud pulmonic sound Palpable pulsations in 2 nd space Right ventricular impulse Prominent <i>a</i> wave in JVP Tricuspid regurgitation Right ventricular third or fourth heart sound Right sided Austin Flint murmur Evidence of mitral stenosis, Preexisting left to right shunt or Eisenmenger syndrome Primary pulmonary hypertension

second sound and that of pulmonary incompetence starts with the pulmonary component of the second sound. This feature is rarely of use in actual practice because in the clinical setting when pulmonic regurgitation occurs, the second heart sound is either closely split or is single, making it difficult to distinguish one from the other. The organic pulmonic regurgitation due to normal pulmonary arterial pressures starts some time after the second sound because it takes a while for the turbulence to occur (Fig. 24.9).

The features of murmur of pulmonic regurgitation depend upon whether pulmonary arterial hypertension (PAH) is present or not. With pulmonary hypertension, the murmur is long, decrescendo and of high frequency. However,

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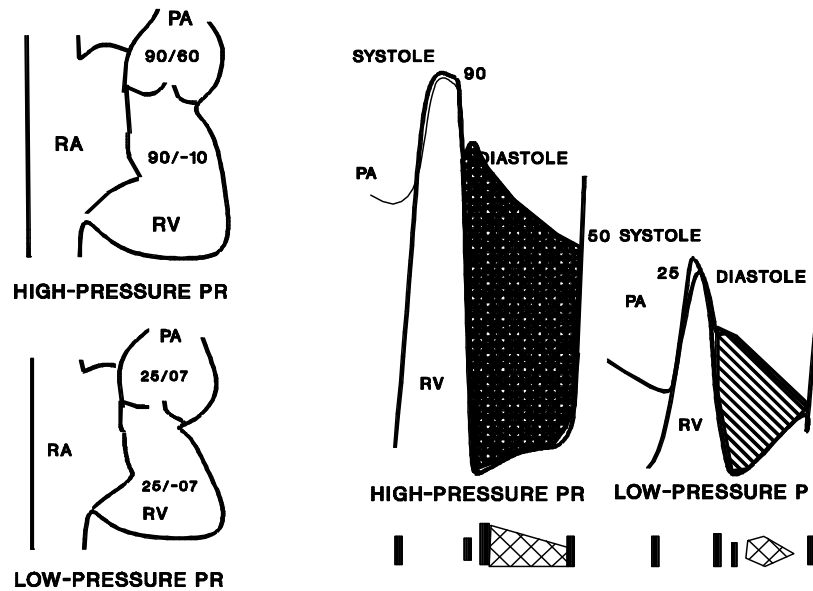


Fig. 24.9: Mechanism of pulmonary regurgitant murmur

Table 24.18: Features of pulmonary incompetence with normal pulmonary arterial pressures

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Feature	Description
Timing	Mid-diastolic
Length	Short, never pandsystolic
Site of best audibility	Pulmonary area
Character	Low frequency, rumbling
Conduction	Localized to pulmonary area, may be heard along the left sternal border
Relation to physiological act	
Posture	Increases during supine/passive leg raising Decreases with standing
Respiration	Increases with inspiration Decreases with expiration
Associated features	Absence of pulmonary hypertension Diminished or absent pulmonic sound Palpable pulmonary artery Hyperkinetic right ventricular impulse Signs of valvular pulmonic stenosis Evidence of infective endocarditis Tetralogy of Fallot

with normal pulmonary arterial pressures, the murmur is more mid-diastolic and is of either medium or low frequency across the right ventricular outflow with lesser pressure gradient (Table 24.18). This murmur sounds mid-diastolic, and when heard along the left sternal border, may be mistaken for tricuspid stenosis or even mitral stenosis. In the setting of tetralogy of Fallot with absent pulmonary valve, the pulmonic regurgitation is accompanied by a rough ejection systolic murmur due to the annulus narrowing. This combination of murmurs is occasionally mistaken for a continuous murmur of ductus. The to and fro character of the murmur and clear time interval between the systolic and diastolic components help to distinguish one from the other.

Length

The pulmonic regurgitation of pulmonary hypertension may be very short or long and may even be pan diastolic. The length of the murmur reflects the duration of pressure difference between the pulmonary artery and the right ventricle in diastole. In severe pulmonary hypertension where the pulmonary artery diastolic pressures are usually above 50 mmHg, the right ventricle end diastolic pressure may never equalise that in the pulmonary artery. This permits a long murmur. The pulmonic regurgitation of normal pulmonary arterial pressures as a rule is short in duration as the pulmonary artery diastolic pressure is 10–15 mmHg with right ventricle end diastolic pressure of less than 7 mmHg. This small pressure difference is rapidly equalised in the later part of diastole, discouraging the possibility of any murmur.

The length of the murmur also helps in the differential diagnosis of aortic regurgitation from pulmonic regurgitation of pulmonary hypertension in one setting. When there is a long early diastolic murmur with no peripheral signs of aortic regurgitation, pulmonary regurgitation is very likely. This is because a long murmur means more aortic regurgitation, and should have the peripheral signs of aortic run off. A long early diastolic murmur with no peripheral signs of aortic regurgitation for that matter means pulmonic regurgitation.

Site of best audibility

The murmur of pulmonic regurgitation due to pulmonary hypertension is best heard at the pulmonary area and may be heard at the apex when formed by the right ventricle. It is unusual for it to be audible to the right of the sternum. The

murmur of pulmonic regurgitation with no pulmonary hypertension is always localised to the pulmonary area.

Character

The early diastolic murmur of pulmonic regurgitation due to pulmonary hypertension is of high frequency and that of pulmonic regurgitation with no pulmonary hypertension is of low frequency. The frequency of the murmur is a function of the pressure difference between pulmonary artery and right ventricle in diastole. Rarely, the pulmonic regurgitation of pulmonary hypertension may have a combination of frequencies with the rough component dominating. This may, in rare cases, be associated with a diastolic thrill. When the pulmonic regurgitation murmur is rough with a diastolic thrill, it is often mistaken for a systolic murmur of ventricular septal defect or pulmonic stenosis. This type of murmur is more likely in pulmonary hypertension due to patent ductus arteriosus and primary pulmonary hypertension. This error of mistaking it for a systolic thrill and murmur is more common than is realized, because one does not expect a rough early diastolic murmur in pulmonic regurgitation. The rough and long murmurs are automatically considered systolic and are ascribed to either ventricular septal defect or pulmonic stenosis. It is in this setting that one should carefully auscultate to identify the sounds first and by inching the stethoscope from the apex to the base.

Causes of pulmonic regurgitation with no pulmonary hypertension

- Congenital isolated pulmonary regurgitation
- Pulmonic stenosis with dysplastic pulmonary valve
- Tetralogy of Fallot with absent pulmonary valve
- Idiopathic dilatation of pulmonary artery
- Infective endocarditis of pulmonary valve
- After balloon dilatation for pulmonic stenosis
- After surgical valvotomy
- After surgical repair of tetralogy

Relation to a physiological act

The pulmonic regurgitation of pulmonary hypertension has no consistent relationship to respiration. It may increase during inspiration; this helps to distinguish it from aortic regurgitation. The murmur fails to increase during

inspiration in a significant number of patients and is related to severe pulmonary hypertension and possibly right ventricular failure, which prevent further increases in cardiac output. In the setting of severe pulmonary hypertension, the hangout intervals on the pulmonic side approximate with that of systemic circulation and respiration may fail to alter it. Significant inspiratory increase in the murmur is more often a feature of pulmonic regurgitation of no pulmonary hypertension. As the murmur of tricuspid stenosis may occasionally be heard nearer the left third space, it may be confused for pulmonic regurgitation. A dramatic increase with inspiration is more likely with tricuspid stenosis than with pulmonic regurgitation. Isometric hand grip and vasopressors increase the murmur of aortic regurgitation and do not influence pulmonic regurgitation.

Accompanying features

Signs of pulmonary hypertension: Severe pulmonary hypertension is a rule with the Graham Steell murmur. The pulmonic sound is severely accentuated and is palpable. In rare cases, the pulmonic sound may not be very loud or may even be interpreted as normal in intensity. This is more likely to happen in patients with primary pulmonary hypertension with right ventricular failure and low cardiac output. With a reduced flow across the pulmonary valve, the valve excursion is limited and the subsequent pulmonary valve closure will be less intense.

Other auscultatory signs: The high frequency murmur of tricuspid regurgitation is common due to high right ventricle pressures, dilated tricuspid annulus, dilated and dysfunctioning right ventricle. A right sided Austin Flint murmur may occur and is related to the pulmonic regurgitation jet preventing adequate opening of tricuspid valve causing relative tricuspid stenosis. A right sided fourth heart sound and third heart sound may occur. A constant pulmonary vascular ejection click is common. It is constant because the right ventricle end diastolic pressures can never exceed the pulmonary artery diastolic pressures in severe pulmonary hypertension. This relationship between pulmonary artery diastolic pressure and right ventricle end diastolic pressure prevents premature opening of the pulmonary valve. The non-pulmonary hypertensive pulmonic regurgitation is accompanied by normal, diminished or absent pulmonic component of second heart sound. The right ventricle impulse is hyperkinetic in a purely volume loaded right ventricle as in pure pulmonary incompetence due to infective endocarditis or idiopathic

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dilatation of pulmonary artery. The RV impulse is hyperkinetic and sustained, when pulmonic regurgitation is associated with pulmonic stenosis, as in pulmonic stenosis with dysplastic pulmonary valve or tetralogy with absent pulmonary valve.

GRAHAM STEELL MURMUR

The Graham Steell murmur in a patient with pulmonary hypertension suggests that the pulmonary artery pressures are in the systemic or suprasystemic range. In the setting of valvular heart disease, this murmur as a rule indicates underlying severe mitral stenosis, or combined mitral stenosis and regurgitation with dominant mitral stenosis. It rarely occurs with pure mitral regurgitation and almost never occurs with aortic valve disease. In the setting of left to right shunts, this murmur is often suggestive of severe pulmonary hypertension near systemic range and often signifies inoperability. Rarely, this murmur may occur in patients with near systemic or systemic pulmonary artery pressures and significant left to right shunts with hyperkinetic shunt dependent pulmonary hypertension (Table 24.19). Never use this murmur to decide on the operability or inoperability, as a small percentage of patients with this murmur may be operable. In a patient with cyanosis, this murmur generally indicates Eisenmenger syndrome or, rarely, congenital cyanotic heart disease with increased pulmonary blood flow and severe pulmonary hypertension as in transposition of great arteries, double outlet right ventricle, single ventricle, or total anomalous pulmonary venous connection.

The early diastolic murmur of pulmonary incompetence can occur in atrial septal defect without any pulmonary hypertension due to dilated pulmonary artery related to large pulmonary flow.

Table 24.19: Significance of Graham Steell murmur

<i>Clinical setting</i>	<i>Significance</i>
Pulmonary artery pressures	Near systemic, systemic or suprasystemic range
Valvular heart disease	Almost always suggests MS or MS + MR with dominant mitral stenosis. Rheumatic heart disease is most likely
Left to right shunts	Severe PAH (fixed or hyperkinetic) No PAH but dilated PA as in atrial septal defect
Cyanotic patient	Eisenmenger syndrome Cyanotic heart disease with PAH

PULMONIC REGURGITATION WITH NO PULMONARY HYPERTENSION

This type of murmur due to pulmonic regurgitation occurs in a variety of situations. In the setting of valvular pulmonic stenosis, it means a dysplastic pulmonary valve, infective endocarditis, post-pulmonary valvuloplasty either surgically or by balloon valvuloplasty. In cyanotic heart disease, it occurs with tetralogy with absent pulmonary valve, or after surgical correction of tetralogy with outflow resection and patch repair (Table 24.20).

Table 24.20: Significance of pulmonic regurgitation with no pulmonary hypertension

<i>Clinical setting</i>	<i>Significance</i>
Pulmonary stenosis	Localises the lesion to the valve May suggest a dysplastic valve Infective endocarditis Helps to distinguish pulmonic stenosis from VSD and AS After balloon dilatation/surgery
Tetralogy of Fallot	Tetralogy with absent pulmonary valve After surgical correction
Patient with fever/septicemia	Infective endocarditis

It is important to check for the murmur of pulmonic regurgitation in patients with pulmonic stenosis because, a dysplastic valve is not easily amenable to balloon dilatation. Infective endocarditis of the pulmonary valve is often mistaken for bronchopneumonia when the diastolic murmur is not detected. In the differential diagnosis of systolic murmur at the pulmonary area, the low-frequency ‘mid-diastolic murmur’ suggests pulmonary stenosis and rules out ventricular septal defect or aortic stenosis.

25 Continuous Murmurs

There are two components in the definition of a continuous murmur. The first feature is the ability of the murmur to overlap the second heart sound and spill over to diastole with no respect for the boundaries of the cardiac cycle. The second feature is that there is no change in character of the murmur from systole to diastole. The continuous murmur differs from a 'to and fro murmur' in one or both of the features. The murmurs of ventricular septal defect + aortic regurgitation, aortic stenosis + aortic regurgitation, mitral stenosis + mitral regurgitation belong to the category of 'to and fro' murmurs.

Various sites of left to right communication where continuous murmurs are possible: patent ductus arteriosus (PDA) (the most common cause), rupture of sinus of Valsalva (RSOV), systemic arteriovenous (AV) fistula, mammary souffle, aortopulmonary (AP) window, and so on.

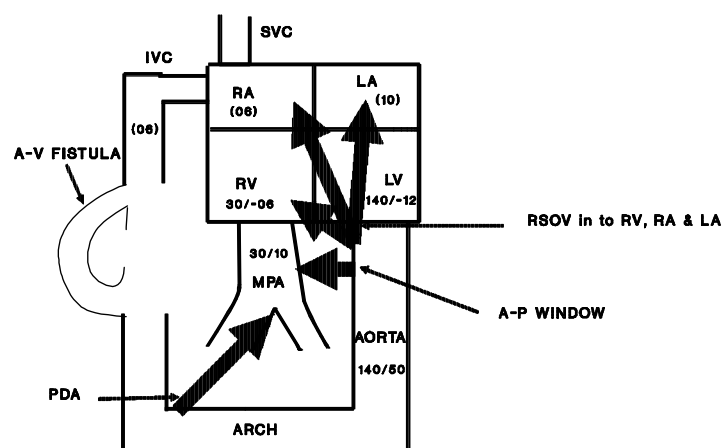


Fig. 25.1: Sites and mechanisms of continuous murmur

Table 25.1: Mechanism of continuous murmurs

<i>Communication between a high and low pressure zone</i>	
	Patent ductus arteriosus
	Aortopulmonary window
	Systemic AV fistula
	Congenital
	<ul style="list-style-type: none"> • Intracranial • Hepatic • Lower and upper limbs
	Acquired
	<ul style="list-style-type: none"> • AV fistula of hemodialysis • Post-trauma • Surgery • Needle puncture • After cardiac catheterization
	Rupture of sinus of Valsalva aneurysm into
	<ul style="list-style-type: none"> • Right atrium • Right ventricle • Left atrium
	Coronary cameral fistula
	Bronchial to pulmonary arterial collaterals in cyanotic heart diseases with diminished pulmonary flow
	<ul style="list-style-type: none"> • Pulmonary atresia • Truncus arteriosus
	Left to right atrial shunt
	<ul style="list-style-type: none"> • Mitral stenosis with PFO/small ASD • Mitral atresia + Small ASD
<i>Narrowing of an artery</i>	
	<ul style="list-style-type: none"> • Coarctation of aorta • Renal artery stenosis • Peripheral pulmonary stenosis • Truncus arteriosus with pulmonary arterial stenosis • Coronary stenosis
<i>Increased velocity of flow with turbulence</i>	
	<ul style="list-style-type: none"> • Truncus arteriosus with pulmonary arterial stenosis • Coronary stenosis • The cervical venous hum • Over the skull in children with anemia • TAPVC supracardiac type • TAPVC infradiaphragmatic/intracardiac
<i>Increased blood flow to an organ</i>	
	<ul style="list-style-type: none"> • Thyrotoxicosis, hypernephroma, hepatoma • Thyroid in thyrotoxicosis • 'Tumour blush' in hepatoma • Hypernephroma • Mammary souffle • Hemangioma

CONTINUOUS MURMURS

The murmur may start any time during systole but should overlap and occupy a varying duration of the diastole. The murmur need not occupy the whole of systole and diastole, to fulfill the definition. Continuous murmurs occur due to a variety of mechanisms. The common mechanisms and the clinical examples are given in Table 25.1.

PATENT DUCTUS ARTERIOSUS

Patent ductus arteriosus is a communication between the proximal descending thoracic aorta 2–10 mm beyond the origin of the left subclavian artery to the origin of the left pulmonary artery from the pulmonary trunk. It is usually 5–10 mm long with a wide aortic orifice and a narrow pulmonary orifice.

At birth, the ductus arteriosus is patent and resembles a muscular artery. Postnatally, closure occurs in two stages.

The first stage is termed as *functional closure*. It is complete 10–15 hours after birth in full term infants and is due to contraction of the smooth muscle in the media of the ductal wall which produces shortening and increase in wall thickness. This functional closure is helped by approximation of the intimal cushions.

The second stage is called *anatomical closure* and is completed within 2–3 weeks after birth. Diffuse proliferation of the intima occurs; this may be associated with necrosis of the inner layer of the media and hemorrhage into the wall. Small thrombi may occur within the lumen. This results in permanent closure of the lumen and results in the formation of a ligamentum arteriosum. The closure most commonly begins at the pulmonary end and may remain open at the aortic end resulting in aortic ampulla. The ductus is completely closed in 88 per cent of children by 8 weeks of age.

Other terms are:

Prolonged patency of ductus: When the process of closure is delayed.

Persistent patency of ductus: When closure fails to occur ultimately.

The factors that influence ductal closure are:

- Release of vasoactive substances (Acetyl choline, bradykinin, endogenous catecholamines)
- Variations in pH
- Oxygen tension (Increasing tension closing the ductus)

- Prostaglandins (PGE_1 , PGE_2)
- Prostacyclin (PGI_2)

The rise in oxygen tension constricts the ductus, and the prostaglandins relax it keeping it patent. The ductus is hypersensitive to oxygen tension in mature fetus but is more sensitive to prostaglandins in immature fetus. These factors are responsible for prolonged patency of ductus in premature infants, especially with respiratory distress.

In 1900, Gibson described the diagnostic continuous murmur of ductus. As depicted in Fig. 25.2, there is a continuous pressure difference between the aorta and pulmonary artery resulting in a continuous murmur. However, if pulmonary hypertension develops, there may not be any diastolic gradient resulting in isolated systolic murmur.

Table 25.2: Continuous murmur of patent ductus arteriosus (Gibson's murmur)

<i>Feature</i>	<i>Description</i>
Site of best audibility	Pulmonary area (infraclavicular, left 2 nd space, 3 rd space)
Timing	Continuous murmur with a late systolic accentuation with maximal intensity nearer to second heart sound, and tapers off in diastole
Length	Starts a while after first heart sound reaches a peak nearer to second heart sound and tapers in diastole. The diastolic component is shorter
Character of murmur	Machinery in quality with a combination of high and low frequencies.
Conduction	The systolic component may be widely audible including the suprasternal notch but the diastolic component is localized to the pulmonary area.
<i>Relation to physiological act</i>	
Respiration	May be better heard during expiration
Isometric hand grip	Increases in intensity and duration
Vasopressors	Increases in intensity and duration
Valsalva	Decreases or disappears
<i>Accompanying features</i>	
	High volume or collapsing pulse
	Left ventricular or biventricular enlargement
	Second heart sound is muffled; or audible pulmonic sound if PAH
	Left ventricular third heart sound
	Mid-diastolic murmur at apex

CONTINUOUS MURMURS

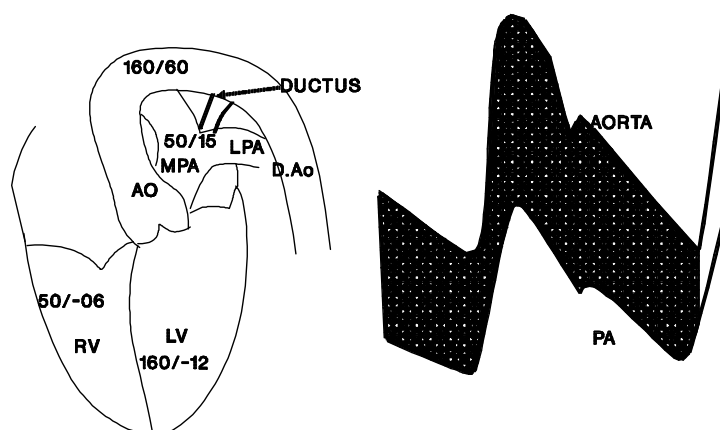


Fig. 25.2: The mechanism of continuous murmur of patent ductus arteriosus

With a continuous murmur typical of ductus, the diagnosis is correct almost all the time. However, once the murmur is atypical, alternative causes for a continuous murmur should be looked for.

Evaluation

In the evaluation of the continuous murmur of PDA, one should ask the following questions.

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- Is it ductus or a condition simulating it?
- If it is ductus, is it isolated, compensatory, or complicated?
- If it is isolated ductus, what size is it?
- What is the size of shunt?
- Is there pulmonary hypertension; if so, what degree?
- Is the pulmonary hypertension hyperkinetic or fixed?
- Are there any associated defects?

A systematic analysis of various features of the murmur will answer most of the questions.

Site of best audibility

The murmur of patent ductus arteriosus is best audible at the pulmonary area and the systolic component is more widely audible at the lower left sternal border, apex, aortic area and over the suprasternal notch. An accompanying thrill is common and is often felt over the suprasternal notch. A thrill over the suprasternal notch is not a feature of ventricular septal defect. Though the systolic component

is widely audible, the diastolic component is restricted to the pulmonary area. If the diastolic component is clearly heard at the apex, ventricular septal defect + aortic regurgitation, or some other cause for a continuous murmur, should be considered. If the murmur of ductus is well heard to the right of the sternum, either an aortopulmonary window, right sided ductus, or a loud venous hum transmitted to the infraclavicular area should be considered.

Timing

The typical murmur of ductus is continuous, peaks in late systole, and tapers off in diastole. Rarely, the murmur may occupy the whole of the systole and diastole. The late systolic murmur is related to the later rise in pressure in descending aorta in relation to systole.

The diastolic component is always shorter than the systolic component. The duration of the murmur is a reflection of the duration of pressure difference between the aorta and pulmonary artery. As pulmonary pressures start getting higher, the diastolic component shortens or disappears first followed by abbreviation or disappearance of systolic murmur. Finally, when systemic and pulmonary pressures become equal, the murmur may be absent altogether. The murmur of ductus may not be continuous when the patent ductus arteriosus is too small (valve like), or too large (equalization of pressures), or when there is pulmonary hypertension (Table 25.3).

Table 25.3: Non-continuous murmurs of patent ductus arteriosus

<i>Cause</i>	<i>Mechanism</i>
Severe pulmonary hypertension	Equalization of pressures
Large ductus	Equalization of pressures
Long, narrow ductus	Valve like mechanism
<i>Associated defects</i>	
Coarctation (preductal)	Lower aortic pressure
Aortic stenosis	Lower aortic pressure + elevated LVEDP leading to PVH and PAH
LV inflow obstruction	PVH - pulmonary hypertension
Mitral regurgitation	PAH - pulmonary hypertension
Peripheral pulmonary stenosis	Central pulmonary hypertension
Ventricular septal defect (large)	Equalization of pressures
D-transposition	Pulmonary hypertension/posteriorly located PA
Infancy	High pulmonary vascular resistance

In very young infants, the diastolic component may be very short or absent, making diagnosis difficult. The murmur is often interpreted as pansystolic and a diagnosis of ventricular septal defect is often made. Careful auscultation with the bell of the stethoscope may bring out the diastolic component of the murmur. Even if this is absent, a high volume pulse with a ventricular septal defect should raise the possibility of patent ductus arteriosus mistaken for ventricular septal defect or associated patent ductus arteriosus with ventricular septal defect. The early diastolic murmur of pulmonary regurgitation of pulmonary hypertension may be pandiastolic occasionally, and when added to the accompanying pulmonary ejection systolic murmur, gives an impression of a continuous murmur. However, the diastolic component being louder than the systolic, the change in character of the murmur from systole to diastole, and the very long duration of diastolic murmur, all rule against a ductus.

Character

The murmur of patent ductus arteriosus is machinery in quality and a thrill is common. It has a combination of frequencies with added multiple clicking sounds. Though the intensity of the diastolic component is less, the quality of both components is the same. If there is any change in the character of murmur between the systolic and diastolic components, ductus is unlikely and ventricular septal defect with aortic regurgitation or some other lesion is likely.

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Relation to a physiological act

The murmur of patent ductus arteriosus either fails to change with respiration nor may increase during expiration. Maneuvers which increase systemic vascular resistance or aortic pressure, like isometric hand grip or vasopressors increase the murmur or may bring out the diastolic component when it is not heard earlier.

Accompanying features

These features help to look for the murmur when not detected in the initial evaluation and also help in distinguishing patent ductus arteriosus from other conditions simulating it.

a) *Cyanosis*: Central cyanosis with a continuous murmur rules out ductus as an isolated lesion, or may suggest another cause for a continuous murmur like bronchopulmonary collaterals.

b) *The high volume or collapsing pulse:* The high volume pulse is the only clue to the underlying patent ductus arteriosus when the murmur is non-continuous. When a diagnosis of left to right shunt is made, a high volume pulse should be used as clue to the ductus as an isolated lesion or in combination with other shunts. The high volume pulse is not diagnostic of ductus because it may occur in any condition with a continuous murmur and aortic run off.

c) *Ventricular enlargement:* Left ventricular enlargement is common with patent ductus arteriosus of any significant size and shunt. Right ventricular enlargement occurs only when pulmonary arterial hypertension sets in. If right ventricular enlargement occurs without signs of pulmonary hypertension, a diagnosis of ductus is unlikely and some other condition like rupture of sinus of Valsalva (RSOV) into the right atrium or right ventricle is likely. This is because the shunt in ductus does not involve the right ventricle, and right ventricle enlargement occurs only after pulmonary hypertension sets in. On the other hand, in rupture of sinus of Valsalva, the aortic run off is directly in to the right heart chambers and right ventricle enlargement of some degree is a rule.

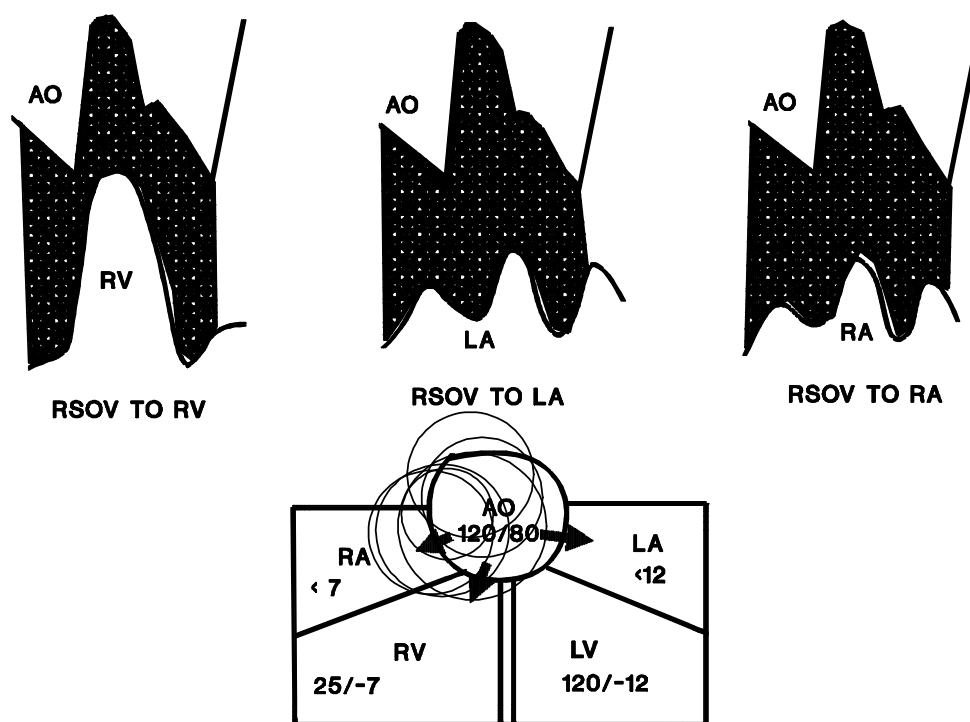
d) *Mid-diastolic murmur at apex:* This murmur means that the shunt is more than 2:1 in a patent ductus arteriosus, and is a reliable sign of operability in spite of pulmonary hypertension. In the presence of pulmonary hypertension if this murmur is audible, the pulmonary hypertension is likely to be hyperkinetic or flow related, in contrast to fixed pulmonary hypertension which is resistance related with pulmonary vascular disease.

RUPTURE OF SINUS OF VALSALVA ANEURYSM

The congenital aneurysms of the sinuses of Valsalva is thinning of the wall of the aortic sinus just above the annulus at the leaflet hinge. Absence of normal elastic and muscular tissue may be responsible. The weakened area gives way under the high aortic pressure to form an aneurysm like a 'wind sock'. This may rupture in an adjacent low pressure cardiac chamber (either the right ventricle or right atrium). The aneurysms arising from the right sinus are most common.

The nature of the continuous murmur in this condition depends on two features: one is the chamber into which the rupture occurs, and the other, the pressure in the receiving chamber. The common site of rupture is either into the right ventricle or right atrium. Rupture into left sided chambers is extremely rare.

CONTINUOUS MURMURS



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Fig. 25.3: Mechanisms of murmur of rupture of sinus of Valsalva. Note that pressure tracings of the aorta are superimposed on the chambers into which the RSOV is opening. Continuous pressure difference leads to generation of a continuous murmur.

The character, location and length of the murmur depend upon the site of rupture. Rupture into the atria always results in a continuous murmur; whereas, rupture into the right ventricle may modify the murmur due to systolic narrowing of the tract and elevation of right ventricle systolic pressure due to pulmonary hypertension. However, the diastolic pressure difference always exists.

The murmur is heard lower along the left sternal border than in PDA and is more superficial with a very prominent diastolic thrill. The description of purring of a cat is most applicable to the thrill accompanying this murmur. As described earlier, the most common murmur is continuous and usually occupies most of the cardiac cycle; it is generally longer than the murmur of ductus.

From the information in Table 25.5, one can understand why the classic murmur of rupture of sinus of Valsalva may not be present in all the patients.

Table 25.4: Murmur of rupture of sinus of Valsalva into right heart

<i>Feature</i>	<i>Description</i>
Site of best audibility	Left sternal border 3 rd - 4 th spaces
Timing	Continuous murmur (CM)
Grade	Almost always grade 4/6 or may be 5/6 and very superficial
Length	Very long almost like a pansystolic plus pandiastolic
Character	Rough with combination of high and low frequencies
Conduction	No selective conduction but is often heard to the right of sternum. Diastolic component is usually not heard at apex
<i>Relation to physiologic act</i>	
Respiration	No significant change as the murmur is very loud (> grade 4/6) and the pressure difference is large in either phase of cardiac cycle
Isometric hand grip	
Vasopressors	
<i>Accompanying features</i>	
High volume or collapsing pulse	Confirms aortic run off as the cause of continuous murmur
Normal intensity S1	First sound is diminished or even absent VSD + AR (severe), which simulates rupture of sinus of Valsalva
Elevated JVP	Supports rupture of sinus of Valsalva into right heart
Tricuspid diastolic murmur	Rupture of sinus of Valsalva into RA
Evidence of large shunt in chest radiograph	Ventricular septal defect with aortic regurgitation is unlikely/rupture of sinus of Valsalva is likely

Table 25.5: Determinants of the murmur of rupture of sinus of Valsalva

<i>Determinant</i>	<i>Nature of murmur</i>
Site of rupture	
Right atrium	Always continuous/longest of murmurs
Right ventricle	Continuous murmur/may vary with right ventricle pressure
Left atrium (rare)	Continuous murmur
Left ventricle (rare)	Early diastolic murmur
Pulmonary arterial pressure	PAH with high RV systolic pressure may shorten or abolish the systolic murmur with persistence of 'early diastolic murmur'
RV outflow obstruction	Similar as above but the ESM of outflow obstruction may be added resulting in a to and fro murmur
Associated VSD	The rough pansystolic murmur of VSD may overshadow the systolic component of continuous murmur giving an appearance of to and fro murmur

CONTINUOUS MURMURS

Table 25.6: Different subsets of murmur of rupture of sinus of Valsalva

<i>Condition</i>	<i>Type of murmur</i>
Rupture in right atrium	Always a continuous murmur
Rupture in right ventricle	Continuous murmur
Pulmonary hypertension (severe)	No systolic murmur/to and fro murmur
RV outflow obstruction (severe)	To and fro murmur (rough ESM + EDM)
Associated VSD	To and fro murmur (rough pansystolic + EDM)

The diastolic component of the murmur of rupture of sinus of Valsalva is generally not audible at the apex, as the aortic run off is directed in to one of the right sided chambers. It is often clearly audible to the right of sternum. On the other hand, the diastolic component of the murmur of ventricular septal defect + aortic regurgitation is usually audible at the apex, as the left ventricle receives the aortic run-off in aortic regurgitation.

MULTIPLE LESIONS

Assessment is easier in an isolated lesion than in a combined lesion or a combination of lesions. The term *combined lesion* is applied to the presence of stenosis and regurgitation in the same valve (for example, mitral stenosis and regurgitation) and the term *combination of lesions* is applied in a broader sense, namely to the presence of any other lesion in the cardiovascular system (mitral stenosis and aortic regurgitation or coarctation of aorta with aortic stenosis).

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Combined lesions are discussed under the following subsets.

- Valvular lesions
 - Combined stenosis and regurgitation
 - Mitral stenosis and mitral regurgitation
 - Aortic stenosis and regurgitation
 - Tricuspid stenosis and regurgitation
- Combination of one valve lesion with another valve
 - Mitral valve disease with aortic/tricuspid valve lesions
- Congenital heart disease
 - Coexistence of more than one lesion
 - Coexistence of valvular lesion with congenital heart disease

- Coexistence of coronary artery disease with valvular or congenital heart defect
- Coexistence of cardiomyopathy with valvular or congenital defect

COMBINED LESIONS IN VALVULAR HEART DISEASE

As a general principle, any regurgitation adds to the amount of blood that has to move across the valve. All the features of stenosis are exaggerated as a rule in combined mitral stenosis and regurgitation.

VENOUS HUM

The normal flow of blood across normal veins in the neck is noiseless. However, when there is increased velocity of flow (hyperkinetic states such as thyrotoxicosis) or diminished viscosity of blood (as in anemia) there is a continuous bruit over the neck veins called the venous hum (Fig. 25.4). In the supine position, with the veins distended there is little or no turbulence, and therefore no hum is heard.

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In a sitting patient, with the bell of the stethoscope lightly applied at the base of the neck in between the two heads of the sternomastoid, the venous hum can be heard as a continuous murmur. The hum disappears when the venous flow is interrupted by applying pressure above the stethoscope. The venous hum is heard in the majority of children and in pregnant women. It is generally abnormal in other adults. It occurs in hyperkinetic circulatory states like anemia or thyrotoxicosis. It also occurs in intracranial or head and neck arteriovenous fistulas. If a venous hum is heard in an adult in the absence of anemia, one should check for thyrotoxicosis. In a child with congestive heart failure and a high volume arterial pulse, one should look for a bruit over the head and a venous hum in the neck to rule out intracranial AV fistula.

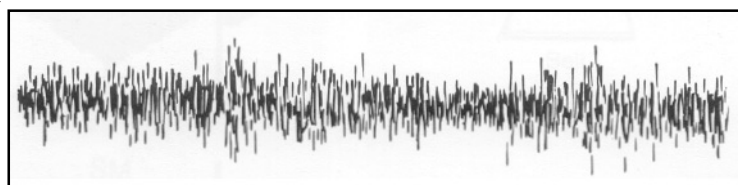


Fig. 25.4: Venous hum

CONTINUOUS MURMURS

Causes

Physiological

- Children
- Pregnancy
- Normal adults (rare)

Pathological

- Beri-beri
- Intracranial AV fistula
- Compression of jugular vein by fascia, or bony structures in the neck

An audible venous hum in the presence of congestive heart failure should suggest the possibility of hyperkinetic states like severe anemia or thyrotoxicosis with heart failure. Intracranial AV fistula is a rare but important cause of heart failure in infancy. A cervical venous hum with high volume arterial pulse and bruit over the skull suggest the diagnosis.

MAMMARY SOUFFLE

This is less common than the venous hum and is heard over the breast during later pregnancy and early postpartum period in lactating women (souffle = ‘puff’

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Table 25.7: Mammary souffle

<i>Feature</i>	<i>Description</i>
Mechanism	Increased blood flow to breast
Timing and length	Continuous, late systolic, systolic component loudest, diastolic component shorter or may be absent
Site of audibility	Over the breast on either side or both sides, 2 nd or 3 rd right or left spaces, no selective conduction
Grade	Always less than grade 3/6
Character	Medium to high frequency but not musical
<i>Response to physiological manoeuvres</i>	
Respiration	No significant change
Pressure over the stethoscope	Light pressure accentuates, firm pressure obliterates
Distal pressure	Obliterates
Valsalva maneuver	No influence
<i>Accompanying features</i>	Advanced pregnancy/lactating mother

in French). The murmur most probably is arterial in origin and is a reflection of increased blood flow to the lactating breast tissue (Table 25.7).

The arterial origin is supported by its delayed onset, systolic accentuation, and persistence of the murmur during the straining phase of Valsalava.

The mammary souffle should be distinguished from patent ductus arteriosus and chest wall arteriovenous fistula. Obliteration by distal compression excludes ductus. The disappearance after stopping lactation is helpful in excluding arteriovenous fistula.

26 The Pericardial Rub

The pericardial rub is a hallmark of pericardial inflammation. It is caused by the parietal and visceral surfaces of pericardium moving against each other. It is uniphasic, biphasic or triphasic in timing. The three components when heard are related to the movement of the heart during ventricular systole, atrial systole (fourth sound time), and rapid ventricular filling phase (third sound time). The most commonly audible component is the one during ventricular systole; the atrial systolic component is the next in frequency and the rapid filling component is the least commonly audible.

The ‘to and fro rub’ is the most common and is due to the ventricular systolic and the atrial systolic components. The three component rub is audible in less than half the cases. The single component rub is very rare and is more likely in the

Table 26.1: Features and description of the pericardial rub

<i>Feature</i>	<i>Description</i>
Location	Left sternal border
Radiation	Pleuropericardial rubs are heard at apex
Timing	Uniphasic, biphasic or triphasic
Character	Superficial, scratchy, or musical (low- or high-frequency)
<i>Relation to maneuvers</i>	
Respiration	Variable, but is usually better heard with held inspiration
Sitting, leaning forward	Better heard
<i>Accompanying features</i>	
Fever, chest pain	Acute pericarditis
Acute MI	Post-MI pericarditis
Uremia	Uremic pericarditis
Elevated JVP, paradoxus, shock	Cardiac tamponade
Acute rheumatic fever	Rheumatic pancarditis

setting of atrial fibrillation (lack of atrial contraction) or the resolving stage of pericarditis. The rub is best heard over the second and third left interspaces over the 'bare area' of the heart. It is best heard in a sitting patient, leaning forward; the diaphragm of the stethoscope should be pressed close to the chest wall. Respiration has a variable influence on the rub. The rub can occasionally be evanescent, and be present in one examination and then surprisingly absent after a few hours. Unlike a pleural rub, which is absent in the presence of a large pleural effusion, a pericardial rub can be present even with large pericardial effusion and tamponade.

Differential diagnosis

The pericardial rub has to be carefully differentiated from the following:

- Systolic murmur (single component rub)
- To and fro murmurs (biphasic rub)
- Early diastolic murmur of aortic regurgitation
- Continuous murmur
- Artifact
- Hamman's sign (mediastinal emphysema)
- Ebstein's anomaly of tricuspid valve
- Tricuspid stenosis
- Right atrial tumour producing RV inflow obstruction
- Means-Lerman scratch in thyrotoxicosis

When very loud and long, the pericardial rub may be mistaken for a continuous murmur. Uniphasic rubs confined to one phase of the cardiac cycle are often mistaken for murmurs. Significant change with pressure on the stethoscope, posture, and localization to the left sternal border help in the differential diagnosis. Expected alterations with physiological maneuvers also help in the recognition of error. Recognition of pericarditis in various clinical situations alters the management and outcome significantly. As the diagnosis of pericarditis is often missed, one must learn to check for this in various clinical settings (Table 26.2).

Pyogenic pericarditis as a cause of cardiac tamponade is often missed; unfortunately it is nearly always fatal. For this reason, in all patients presenting with fever (even if it is of one day duration) or any evidence of septic focus one must check for the pericardial rub. In patients with fever of unknown origin, apart from looking for this sign in the initial evaluation, it must be looked for in all reevaluations as it may appear later in the clinical course of some patients with

THE PERICARDIAL RUB

Table 26.2: When to check for pericardial rub

<i>Clinical setting</i>	<i>Implications</i>
Fever, chest pain or septic focus	Infective pericarditis, pyogenic pericarditis can be rapidly fatal and is often missed
Fever of unknown origin	Tuberculosis
All patients in shock, 'heart failure'	Cardiac tamponade
All patients with acute chest pain	Post-MI pericarditis/primary pericarditis
Acute rheumatic fever/heart disease	Pancarditis/indication for steroids
Infective endocarditis vs rheumatic carditis	Rheumatic carditis is likely
Infective endocarditis	Ring abscess or myocardial abscess
Chronic renal failure on hemodialysis	Cardiac tamponade during dialysis
Amebic liver abscess	Abscess communicating with pericardium
Chest pain, fever, after cardiac surgery	Post-cardiotomy pericarditis
Any patient with known malignancy	Pericardial involvement

tuberculosis or other chronic granulomatous diseases. In all patients with shock, cardiac tamponade should be ruled out by not only careful clinical examination but also by an echocardiogram.

When patients with established rheumatic heart disease present with fever, it is often not easy to differentiate infective endocarditis from recurrence of rheumatic fever. Pericarditis favours the diagnosis of rheumatic fever. In patients with chronic renal failure with pericarditis on hemodialysis, hypotension during dialysis may mean relative hypovolemia precipitating cardiac tamponade even at low pericardial pressures.

If the pericardial rub appears along with the beginning of chest pain, primary pericarditis as a cause of chest pain is likely. On the other hand, if the rub is heard 24–48 hours after the onset of chest pain, post-MI pericarditis is likely. As the ECG may fail to show diagnostic changes or even may show features shared by both conditions, detection of pericardial rub is critical to proper diagnosis and management of these patients.

In acute MI, the pericardial rub may appear any time after the first day to 6 weeks after MI, and is a more sensitive indicator of pericarditis than even the echocardiogram. Pericarditis in this setting indicates a transmural myocardial infarction and is a contraindication to anticoagulant therapy. It gives a clue to the

cause of pain following myocardial infarction. The pain of pericarditis is promptly relieved by steroids.

Significance of pericardial friction rub after acute MI

- Indicates a transmural myocardial infarction
- Contraindication to anticoagulant therapy
- Cause of persistent pain following myocardial infarction
- May be the cause for post myocardial infarction fever
- Prompt relief with steroids

It is for this reason, that all patients with suspected or diagnosed acute ischemic syndromes should have careful auscultation to rule out pericardial rub.

27 Approach to Auscultation

Auscultation, like any other skill, requires practice. Just like a tennis player does wall practice, as a student, one must learn to practice auscultation in otherwise normal hearts. Auscultate your own heart or of patients in the wards without heart disease to have an idea of the sounds of a normal heart. Patients with typical disorders like mitral stenosis should be similarly auscultated systematically. For, example, when the diastolic murmur of mitral stenosis is best heard at the apex, slowly move away from the apex concentrating on the murmur. Though faint, the murmur is audible. This is the type of murmur one may encounter in a patient with 'silent' mitral stenosis. Practise similarly with the opening snap. Once the opening snap is appreciated at the site of best audibility, namely internal to the apex, by slowly moving away one can appreciate the faint opening snap even to the right of the sternum. This type of faint opening snap may be the only sign of mitral stenosis in some situations.

DESCRIPTION OF AUSCULTATORY EVENTS

Note the heart rate

The heart rate influences the duration of various events in cardiac cycle. For example, with the same degree of mitral stenosis, bradycardia with leisurely diastole abbreviates the murmur, and tachycardia with a hurried diastole prolongs the murmur. The S2-OS interval is longer with bradycardia and shorter with tachycardia.

Note the type of chest

Chest wall thickness influences the intensity of sounds and murmurs. Chest

deformities may produce heart murmurs or exaggerate them. Particularly make note of pectus excavatum, straight back and kyphoscoliosis.

Identify first and second sounds (and systole and diastole)

This is best done by starting at the pulmonary area where the second heart sound is louder than the first heart sound. The carotid pulse and apical impulse may also be used for the same purpose but are probably less reliable. In case of difficulty, more than one method should be used. When the heart rate is rapid, the carotid method of identifying can be misleading as the carotid impulse is not as sharp an event as that of first heart sound or second heart sound. In some situations, the systole and diastole may be confused with each other, even by experienced auscultators:

- When the rhythm is irregular or the rate is too fast or too slow
- When both first heart sound and second heart sound are diminished or absent
- When an additional sound is unusually loud, such as the third heart sound at the apex in mitral regurgitation
- Acute aortic regurgitation with diminished or absent first heart sound and second heart sound with loud third heart sound mistaken for first heart sound
- When a loud pericardial knock is heard as in constriction with systolic retraction of the cardiac impulse. The pericardial knock is mistaken for the first heart sound
- When a murmur has unexpected features. As in aortic regurgitation with a thrill and rough quality of the murmur or pulmonary regurgitation with rough quality and a thrill.

Describe the first and second heart sound

First sound

Intensity

Split

Constancy or variability

Second sound

Split

Intensity of both components

Describe additional sounds

APPROACH TO AUSCULTATION

Third sound

- Right or left sided
- Physiological or pathological
- Early or late

Fourth sound

- Physiological or pathological
- Right or left sided
- Opening snap
- Mitral or tricuspid
- S2–OS interval

Ejection click

- Right or left sided
- Valvular or vascular
- If valvular, constant (aortic) or variable (pulmonic)

Non-ejection click

- Left or right sided
- Single or multiple

Describe murmurs

Individual murmurs have already been discussed in Chapters 22 to 25.

Describe presence or absence of pericardial rub

If an auscultatory finding is expected but is not detected, mention it specifically. This has significance; for instance the absence of ejection click in a child with aortic stenosis may mean subvalvular aortic stenosis. As a student if you cannot detect an expected event, mention clearly that you checked for an opening snap but could not hear it.

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